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THE WRITTEN PAPER OF THE FINAL M.B., B.Ch. EXAMINATION IN MEDICINE

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For many years I have shared the misgivings of others that the written paper in Medicine of a final qualifying medical examination affords a poor indication of the candidate's knowledge and ability to think, correlates badly with the results of the examination as a whole, including the clinical and oral sections, and does little more than separate a class into a very few bad candidates, still fewer very good candidates, and a very large group with marks bunched closely together around an indeterminate mean mark.

At the University of the Witwatersrand, written papers were abolished in the final examination in Medicine, Surgery, and Obstetrics and Gynaecology in 1952, and a statutory written examination was introduced at the end of the 5th year of study. In November 1959, written examinations were reintroduced into the final examination on account of curriculum arrangements. Certain subjects such as Gynaecology and Paediatrics were taught to a large extent in the 6th year and written papers at the end of the 5th year could not take this 6th-year teaching into account. The examination in these two subjects at the end of the 6th year was clinical and oral only, as it was in all subjects of the final examination.

In Medicine, irrespective of whether or not there is a written examination, each candidate has a clinical examination lasting 2 - 3 hours, during which he sees 3 examiners independently and examines and discusses 4 - 8 cases, some 'major' and some 'minor'. There is no exact timing system. The oral examination consists of 2 periods of 12 minutes each, during which he sees a different group of 3 examiners, 1 alone and the other 2 together. Before the written papers were abolished, the clinical and orals were regarded as carrying far more weight than the written papers, and this applies now, too, since the reintroduction of written papers. The examiners are not aware of the marks awarded for any section of the examination until a candidate has completed the whole examination.

The Department of Medicine was impressed by the advantages accruing to a final examination with no written papers, and was not anxious to reintroduce papers into the final examination, but abided by the majority decision of the Faculty of Medicine. No falling off in standards was observed by internal or external examiners during the period of abolition of written papers in the final examination.

When written papers were reintroduced to the final examination in November 1959, the Department of Medicine decided to alter the character of the written examination by introducing papers of 'objective' type additional to the papers of conventional 'subjective' or 'essay' type. The answer to the question of *purely* objective type can be only right or wrong, and marking can in fact be achieved by a punch-card system. Take, for example, the question, 'The action of insulin is to raise/lower the blood sugar. Delete which is false'. To this the answer can be only one of two—either the word 'raise' or the word 'lower' has to be deleted by the candidate. An example of the conventional essay type of question is, 'Describe the causes, clinical features and differential diagnosis of acute glomerular nephritis'. Such a question may be answered well or badly in many ways, and the subjective feelings of the examiner are prominent in the marking.

Two questions were introduced in General Medicine that were dominantly but not entirely objective. The remainder of the written questions were of the conventional essay type. There were thus 2 questions of objective type and 2 of essay type in General Medicine, 2 of essay type in Paediatrics, 1 in Dermatology and 1 in Fevers.

The two questions of objective type were as follows:

Question 3

(*a*) Name the bedside tests of assistance in the investigation of a patient with purpura.
(*b*) Define the term 'haemoglobinopathy'. Give three examples.
(*c*) List *three* drugs which can produce a haemolytic anaemia and indicate *briefly* how they exert such an effect.
(*d*) Give the rationale behind the direct Coombs test in not more than five sentences. Name *three* diseases in which the test may be positive.
(*e*) Draw short family trees illustrating the mode of inheritance of (*a*) Haemophilia, (*b*) Congenital spherocytosis.
(*f*) Give a short classification of the lymphomas.
(*g*) *Name* the treatment of choice in each of the following conditions. (No discussion is required): (i) Pernicious anaemia of pregnancy. (ii) Acute lymphatic leukaemia. (iii) Congenital spherocytosis. (iv) Chronic myeloid leukaemia. (v) Idiopathic haemolytic anaemia. (vi) Haemophilia. (vii) Christmas disease. (viii) Paroxysmal nocturnal haemoglobinuria.
(*h*) Indicate briefly the ways in which vitamin-B12 deficiency may develop.
(*i*) List *five* important clinical features that would make you suspect a diagnosis of pernicious anaemia.
(*j*) Define secondary polycythaemia. Give two important causes of this condition.

Question 4

(A) *Insulin*
(*a*) Enumerate three commonly used types of insulin.
(*b*) Record in graphic form their action on the blood sugar.
(B) *Congestive Cardiac Failure*
(*a*) Enumerate the signs of congestive cardiac failure.
(*b*) Discuss any *one* of the signs that you have enumerated.
(C) *Neurological Localization.* State sites of Lesion In:
(i) Subacute combined degeneration of the cord. (ii) Tabes dorsalis. (iii) Progressive muscular atrophy. (iv) Wernicke's encephalopathy. (v) Guillain-Barré syndrome. (vi) Trichlorethylphosphate poisoning. (vii) Lead poisoning. (viii) Diabetic neuropathy. (ix) Jacksonian epilepsy. (x) Freidreich's ataxia.

Each of these objective questions was marked independently by two examiners, neither of whom was aware of the mark awarded by the other. Examiner A set and marked question 3 and the same question was independently marked by examiner B. Examiner C set and marked question 4

and the same question was marked independently also by examiner B.

Two questions of essay type on subjects of common and general interest were selected, to be marked by two examiners independently. These questions were:

Question 1

Describe the causes, symptoms, signs and laboratory diagnosis of obstructive jaundice indicating in each group the contrast or differential diagnosis from hepatocellular jaundice.

Question 5

(*Paediatrics*)

Describe the symptoms, signs, diagnosis, laboratory investigations and treatment of acute rheumatic fever.

Question 1 was set and marked by examiner D and marked independently by examiner C. Question 5 was set and marked by examiner E and marked independently by examiner F.

For the purposes of this comparative study, the marks awarded by the examiners who set and marked the following 3 questions of essay type additional to those referred to above were selected for analysis:

Question 2: set by examiner H

Give (*a*) the essential features for the diagnosis of nephritis; (*b*) the treatment of epilepsy; (*c*) the prognosis of bronchial asthma.

Question 6 (*Paediatrics*): set by examiner E

Discuss the approach to the diagnosis of the causes of vomiting in the first year of life.

Question 7 (*Dermatology*): set by examiner G

Discuss the treatment of: (*a*) the seborrhoeic diseases of the hairy scalp; (*b*) acne vulgaris.

The marks awarded in Dermatology were selected for analysis because it was thought that Dermatology might be a subject that does not receive the same attention and interest of the candidates as subjects in General Medicine and Paediatrics.

In the total final examination in Medicine, including clinicals, orals and written papers, there were 14 failures, each with a mark below 50%; 43 passes, each with a mark of 50% - 59%; 14 exemption passes, each with a mark of 60% - 64%; and 8 second-class passes each with a mark of 65% - 74%.

For each written question under analysis, the mean mark awarded by the examiners was calculated for the candidates falling into each of these above-mentioned 4 groups in the total examination, i.e. fail, ordinary pass, exemption pass, and second-class pass. Thus, for question 3, for example, the mean mark of the 14 candidates who failed the total examination was calculated and found to be 38%, of the 43 ordinary passes 55%, of the 14 exemption passes 60%, and of the 8 second-class passes 68%. The marks of each question were analysed in this way and are tabulated in Table I and set out in graphic form in Fig. 1. From these results the following observations can be made:

TABLE I. MARKS FOR WRITTEN QUESTIONS OF SUBJECTIVE AND OBJECTIVE TYPE RELATED TO RESULT OF TOTAL EXAMINATION IN MEDICINE, WRITTEN, CLINICAL AND ORAL

Result of total examination with numbers in each pass or fail category	*Subjective questions*					*Objective questions*	
	GM 1	*GM* 2	*Derm.*	*Paed.*		*GM* 3	*GM* 4
	D	*H*	*G*	*E*	*E*	*A*	*C*
Fail, 14	52	52	37	52	52	38	43
Pass 50-59%, 43 ..	56	57	44	57	59	55	53
Exemption 60-64%, 14	59	58	47	58	60	60	64
2nd class 65-74%, 8 ..	62	59	49	60	63	66	69

Marks = mean percentage for written questions. See text for method of calculation. GM = General Medicine. Capital letters identify examiners.

1. Only in the two objective written questions did the mean mark for the total examination correlate with the mean mark for individual written questions. In Fig. 1,

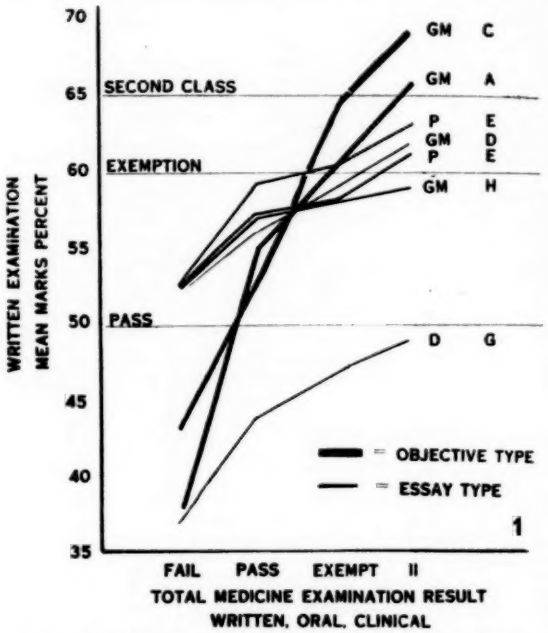

Fig. 1. Correlation of results of written and total examination in Medicine. GM = general medicine. P = paediatrics. D = dermatology. Capital letters identify examiners awarding marks.

the graph of the mean mark of the two objective questions passes through all four of the categories in which the total examination result falls, i.e. from fail to second class.

2. In *all* the subjective questions the graph line of the mean mark ran insipidly through a small range for all categories of the total examination from failed to second-class pass.

3. In the subjective questions of *general* interest, such as jaundice, rheumatic fever, vomiting in infants, nephritis, epilepsy and bronchial asthma, this insipid graph line ran through a range of mean marks of 52% - 63%. None of the mean marks dropped to a fail level or rose to a second-class level.

4. In the subjective question in Dermatology, a subject of more limited interest, the same type of insipid graph line ran entirely below the 50% mark but about parallel to the graph line for the other subjective questions.

In other words, the correlation between marks awarded for individual written questions of objective type and the result of the total examination including clinicals and orals was good, whereas the marks awarded for the written questions of subjective type were indeterminate for fails and for the candidates with the best passes in the total examination.

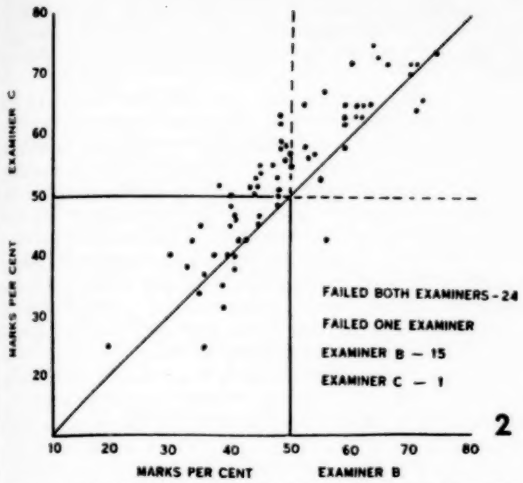

Fig. 2. Marks awarded by 2 independent examiners for written objective question (question 4).

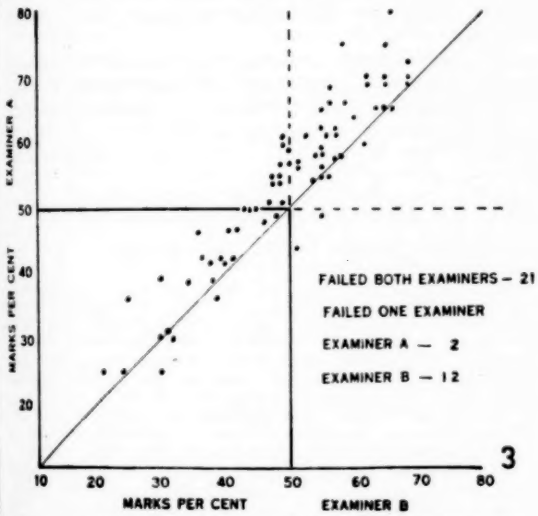

Fig. 3 Marks awarded by 2 independent examiners for written objective question (question 3).

In Figs. 2 - 5 are set out graphically the individual marks awarded by 2 independent examiners marking each of 4 questions, 2 of them objective and 2 subjective. From these diagrams the following observations can be made:

1. In the two objective questions (Figs. 2 and 3) the marks of both examiners covered a wide range from fail to second-class pass with a good scatter and good correlation in spite of individual differences in standards as shown particularly by the numbers failed by both examiners and by a single examiner.

2. In the two subjective questions (Figs. 4 and 5) the marks of both examiners were bunched together in the pass area over a narrow range and with poor scatter. Very few candidates were failed by one or both examiners.

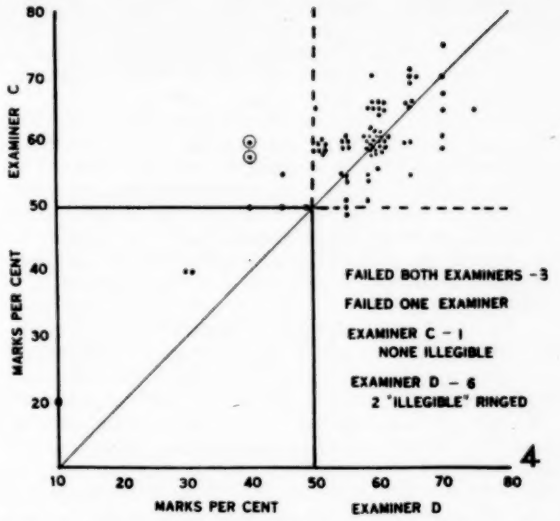

Fig. 4. Marks awarded by 2 independent examiners for written subjective question (question 1).

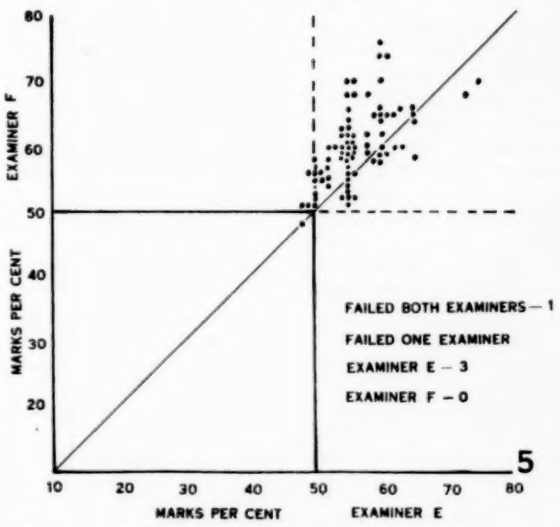

Fig. 5. Marks awarded by 2 independent examiners for written subjective question (question 5).

3. In the objective questions examiner B, who was the second examiner in both, failed considerably more candidates than examiners A and C respectively, who had set and marked the questions and whose marks appear in Table I. Clearly, the factor of the personality of the examiner enters into these questions, although not interfering with the correlation of marks between the two examiners and not interfering with the correlation with the result of the total examination. It was found in discussion that examiner B had made high demands of accuracy. For example, he marked down candidates who failed to record correctly the exact times of fall and rise of blood sugar after injection

of insulins, whereas examiner A gave more credit for graphs of blood-sugar curves that gave a general indication that the candidate knew the comparative differences in action of the three insulins even though the recording of individual times of fall and rise of blood sugars were not accurate.

4. Examiner D gave poor marks on account of 'illegibility' to two candidates, whereas examiner C, marking independently, gave good marks to these same two candidates and did not regard their handwriting as illegible. The surname initials of these two candidates were V and W, suggesting the possibility that examiner D was fatigued as he approached the last papers, and found illegible handwriting that he might well have deciphered had he been in a less fatigued state.

DISCUSSION AND CONCLUSIONS

In spite of the fact that the objective questions set in this examination were not purely objective, in that they contained discussion and essay-type elements, the marks awarded for these questions were far more discriminative than the marks for the subjective questions, and correspond well with the final total result of the examination, including results of the clinical and orals. The marks awarded for the questions of subjective type bore no relationship whatsoever to the results of the total examination.

Correlation of marks awarded by independent examiners was good for the objective questions in spite of the severer marking standards of one of the examiners. This difference in marks did not interfere with the good correlation with the final total result of the examination. Objective questions lend themselves to standardization of answers. In the recent trial no attempt was made beforehand to set the standards of marking of the objective types of question, although this could legitimately have been done and will be done in future.

In the subjective questions, not only did the marks not bear any relation to the final total examination result, but the marks given by two independent examiners bore little relation to each other owing to their all being bunched together in the pass range with very few failures. It is impossible to standardize answers for subjective questions.

The two types of question test different qualities:

The objective question demands accuracy of factual knowledge and at the same time affords the candidate a chance to express himself concisely and clearly either in words, which tests literacy, or by diagram. Without factual knowledge the candidate is bound to fail.

The essay-type question tests literacy and the power of expression, but ignorance of factual knowledge can be concealed by a well-written paper. Furthermore, a great amount of factual knowledge can be presented badly and the candidate be awarded a poor mark.

There would appear to be a place for each type of question in the final medical examination, but the one that bears correlation with the final total examination result is the objective question. The question of essay type is indeterminative.

It might, however, be justifiable to ask, if the objective written question correlates well with the final total result of the examination and the essay-type question correlates not at all, why have written questions?

EXPERIMENTAL NON-DIETARY CIRRHOSIS IN RATS*

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A description was given of a Laennec-type cirrhosis produced in rats by the following method: On the first day of the experiment 2 ml. per 100 g. body-weight of egg yolk diluted 1 : 1½ in isotonic saline was injected intravenously and the left and right renal pedicle tied 4 - 6 hours later. Intravenous egg-yolk injections were then continued every second to fourth day until the rats were killed. During this time they were allowed water and rat cubes *ad lib.*

Although the injections were large they were well-tolerated. They did, however, cause oedema of the paws, redness and some swelling of the ears, and nose scratching. These signs are customarily regarded as evidence of histamine liberation in the rat. Cirrhosis occurred in 15 consecutive experiments when the method outlined was carefully followed, and developed in the surprisingly short time of 21 - 48 days. At autopsy the liver was finely granular and generally slightly enlarged. Microscopical examination confirmed the cirrhosis but showed no evidence of massive, zonal or focal necrosis. Necrotic liver cells were observed, but these occurred as single isolated cells or as small groups of cells at the periphery of the lobule and were most easily found in the earlier stages of the process when fibrous-tissue bands were just beginning to proceed from the portal tracts. A moderate infiltration of haemosiderin-laden histiocytes, plasma cells and eosinophils accompanied the fibrous-tissue response, but fatty change was absent in all cases.

Cirrhosis also developed in 2 of 3 rats in whom unilateral nephrectomy was substituted for unilateral renal-pedicle ligation and in a further 2 rats when 1% salt was put into the drinking water and no renal operation performed. Cirrhosis was absent in 5 animals subjected to unilateral renal pedicle ligation alone, but did develop in 2 of 6 rats given intravenous egg yolk alone. The limited success with egg yolk suggested that increased physiological strain on the kidney acts as an adjuvant in the pathogenesis of this type of cirrhosis, and attention was drawn to the renal lesions that occur in choline deficiency and after carbon tetrachloride poisoning.

Discussion was largely confined to the pathogenesis of the experimental lesion and little attempt made to relate the findings to human pathology, though they might seem applicable to the cirrhosis of porphyria. It was almost certain that some functional overload of the liver occurred after such large intravenous injections of egg yolk, but this did not seem to operate either by producing a relative deficiency of lipotropes or by any known hepatoxic action. The experiments appeared to have a precedent both in Eppinger's early work relating the action of histamine to experimental cirrhosis and also in recent reports from Japan of Laennec cirrhosis following partial ligation of the inferior vena cava and injections of histamine or egg albumin. However, some features of the present experiments as well as preliminary failure to produce cirrhosis with the histamine liberator 48/80 suggested that histamine liberation alone was probably not the most important factor in the production of cirrhosis in the egg-yolk experiments.

Bacterial infection was thought to be a most unlikely explanation of the cirrhosis following egg-yolk injections, but this possibility was only completely excluded in the later experiments.

* Abstract of paper presented at Research Forum, University of Cape Town, 3 February 1960.

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EDITORIAL : VAN DIE REDAKSIE

MALNUTRITION IN CHILDHOOD

'An army marches on its stomach'. This phrase calls to mind the campaigns of bygone days, in which thousands of men, marching into alien territory, were entirely dependent for their progress, their achievements and their very existence on the rations supplied for them. If the food supplied was not adequate for their needs, their progress was slowed, their achievements reduced and many died of famine or disease, against which, in their weakened state, they had little resistance.

In South Africa today, as in many parts of the world, an analogous picture is seen among tens of thousands of infants and young children, whose physical progress is slowed and whose mental achievements are retarded because the food they are given is inadequate for their needs. Many die from frank nutritional disorders such as marasmus, kwashiorkor or rickets, and many from diseases like gastro-enteritis and pneumonia, which should not be fatal, particularly in the one to four years age group, but which are the cause of thousands of non-European deaths in childhood below the age of five years. It is a point of great significance which is not generally recognized that while the infantile mortality rate among non-Europeans is four to six times the rate among European infants, the death rate in the age group one to four among the Coloured population is fifteen times, and among the Bantu thirty times as great as the European death rate at this age. A large part of these deaths are due to the causes mentioned above. The basic reason for this state of affairs lies in lack of essential foodstuffs, particularly protein, in the daily diet of these children. This is due in part to ignorance, but largely to the conditions of poverty into which they are born.

In this issue of the *Journal* we publish an article by Robertson, Hansen and Moodie, who show from the available figures what an enormous loss of infant life there is in this country from gastro-enteritis, and have produced evidence to show that a very large proportion of this is due primarily to malnutrition. There is no doubt that an investigation into infant deaths from pneumonia would produce a similar picture.

Schrimshaw and Béhar[1] have recently pointed out that for every case of kwashiorkor in a population group there are at least a hundred cases of underlying protein malnutrition sometimes referred to as pre-kwashiorkor. These cases can best be recognized by the fact that their weight is below tha expected for their age.

The published vital statistics do not reveal the vast role played by malnutrition in the morbidity and mortality of infants and pre-school children, particularly non-Europeans, in this country. The nutritional factor is frequently not taken into account when the cause of death is certified, or malnutrition is mentioned only as a contributory cause, the terminal disease being given as the primary cause of death. More discerning certification would show a tragic picture of thousands of young children dying annually in the Union because of inadequate feeding.

A dramatic change in child health in Britain has taken place since the war, nutritional disorders and their accompanying diseases having practically disappeared as the result of improvement in social conditions and the provision of sufficient milk for every child at low cost. Although the Union is not a welfare state, much can yet be done within the framework of our local conditions to alleviate the present position.

A tremendous step towards the elimination of malnutrition and its associated diseases would be the widespread distribution of milk or other foods rich in protein to infants and pre-school children at low cost. The local bodies distributing such foods would require assistance in the form of subsidy to achieve this end. As a corollary to this, extension of child welfare services is needed for the instruction of the mothers and supervision of the health of the children. The treatment of gastro-enteritis by doctors in hospitals or elsewhere should be accompanied by advice on nutrition and as early a return to full milk diet as possible. This point is brought out in an article by Bowie in this issue of the *Journal* on the out-patient treatment of infant cases of gastro-enteritis with dehydration. Far too many doctors keep children on inadequate diets while they are ill, thus increasing the malnutrition.

A vast amount of money is being spent on building and running hospitals and out-patient departments, which are all overcrowded with sick children. Improved nutrition in the pre-school years would eliminate a great deal of preventable sickness and death among children, most of whom should never be in need of hospital treatment.

1. Scrimshaw, N. S., and Béhar, M. (1959): Fed. Proc., 18, 82.

HERSTELSALE VIR BEWUSTELOSE PASIËNTE NA OPERASIES

Die probleem van 'n geskikte lokaal wat spesiaal toegerus is as 'n afdeling in hospitale waar bewustelose pasiënte na operasies kan herstel voordat hulle teruggaan na die algemene sale of na hulle kamers, word om verskillende redes al meer dringend en belangrik. Dit sal dus goed wees om aspekte van hierdie probleem hier kortliks te bespreek.

Om mee te begin moet ons dink aan die uitwerking wat die herstel van 'n pasiënt wat onder narkose was, op ander pasiënte in 'n algemene saal het. Dit ly geen twyfel nie dat daar iets vreemds en verontrustends en ontstellends in die hospitaal-situasie geleë is vir die meeste mense. Dit is verstaanbaar dat dit so is en dit is maar net menslik. En die geluide en die ‚doenigheid' wat gewoonlik gepaard gaan met die regkom van bewustelose pasiënte, het soms 'n verskrikkende en angswekkende uitwerking op pasiënte wat lê en wag dat hulle beurt om 'n operasie te kry, aanbreek. Hierdie toestand van sake is sonder enige twyfel baie ongewens. Soos dit ook die geval is met ander vertakkings van die medisyne, is dit noodsaaklik om die welsyn van die ‚hele mens' in gedagte te hou, en om alle maatreëls te tref om die pasiënt

voor en na 'n operasie so gerus moontlik te stel en so gelukkig moontlik te hou.

'n Ander faktor wat die aandag in die jongste tyd op die behoefte aan spesiale herstelafdelings gevestig het, is die algemene tekort aan verpleegsters. Dit is die oorwoë opvatting van narkotiseurs en van verpleegsters wat ondervinding het van hoe sulke afdelings funksioneer, dat die instelling van afdelings vir die herstel van bewustelose pasiënte in hospitale tot 'n groot besparing van arbeidskragte kan lei.

Baie matrones van hospitale het aan die begin gewoonlik besware in hierdie verband omdat hulle reken dat meer personeel benodig sal word. Waar die skemas egter al in werking getree het, is die personeel self gou verwonderd oor hoe hulle so lank daarsonder klaargekom het. Herstelafdelings stel die suster wat in beheer van die algemene afdeling is vry van die verpligting om toe te sien dat daar by elke geval 'n spesiale verpleegster is totdat die geval ten volle van die narkose herstel het, en totdat die gevare van na-operatiewe skok en bloeding verby is.

Herstelafdelings, soos wat ons in gedagte het, behoort by alle hospitale en verpleeginrigtings in gebruik gestel te word en daar behoort voorsorg gemaak te word vir verplegingspersoneel wat spesiaal vir die doel opgelei is. Ook moet die afdelings uitgerus wees met geskikte apparaat vir die bybring van bewustelose pasiënte, soos suigpompe, beddens wat kan oorhel, voorrade van suurstof, ens.

Die Uitvoerende Komitee van die Suid-Afrikaanse Vereniging van Narkotiseurs (M.V.S.A.) het 'n spesiale versoek aan die Federale Raad van die Mediese Vereniging gerig om hul pogings in hierdie verband te ondersteun, en die Federale Raad het dan ook, by geleentheid van sy onlangse sitting in Pretoria (3 - 5 Maart 1960) sy steun hieraan toegesê. Daar is geen twyfel aan nie dat 'n skema soos hierdie die volle steun van die hele mediese professie en die verpleegstersberoep verdien.

THE PROBLEM OF GASTRO-ENTERITIS AND MALNUTRITION IN THE NON-EUROPEAN PRE-SCHOOL CHILD IN SOUTH AFRICA

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Official figures from the South African Bureau of Census and Statistics[1] for individual causes of death in pre-school children indicate that the main cause of death in this age group is gastro-enteritis. The loss of life from this cause is of staggering proportions; it is estimated that more than 11,000 pre-school children die annually from gastro-enteritis in the urban centres alone. Although figures in this paper refer to urban populations a similar state of affairs exists in rural areas, for which statistics are not available.

The gastro-enteritis figures present a challenge of the first magnitude and urgency to the medical and health services of this country. In this paper we have endeavoured to present some detailed statistics of the problem of gastro-enteritis in South Africa, to analyse the causes and effects of the high incidence and mortality, and to emphasize the importance of malnutrition. Practical preventive and therapeutic measures that can be adopted at national and local level to reduce mortality and morbidity rates are set out.

Statistics

Although statistics relating to gastro-enteritis and malnutrition in the country as a whole are incomplete, the total numbers of deaths occurring in urban areas are reported by the local authorities and, from these figures and those published by the Union Health Department, some idea of the total problem can be formed[2,3] (Table I).

TABLE I. DEATHS FROM GASTRO-ENTERITIS, UNION, 1957

	Deaths from gastro-enteritis
European	516*
Asiatic	286*
Coloured	4,750*
Bantu	5,742†
Total recorded	11,294

* As recorded by Union Health Department.[3]
† As recorded by 14 local authorities.[2]

Deaths from gastro-enteritis constitute 2% of all European and 22% of all Coloured deaths.[1] Almost all these deaths occur under 5 years of age. The position in the Union as a whole is shown in Table II,[1] in which the deaths from gastro-enteritis are expressed as a percentage of the total deaths in the same age and racial group. Table III gives similar figures for the City of Cape Town.[4]

TABLE II. DEATH RATES FROM ALL CAUSES FOR VARIOUS AGES AND BY RACE, PERCENTAGE DUE TO GASTRO-ENTERITIS ALONE: UNION, 1956.[1]

Race	*Deaths from all causes*			*Deaths from gastro-enteritis as % of deaths from all causes**		
	per 1,000 live births	*per 1,000 population**				
	0 - 1 yr.	*1 - 4 yrs.*	*5 yrs. & over*	*0 - 1 yr.*	*1 - 4 yrs.*	*5 yrs. & over*
Eur.	31·4	1·5	8·9	10·3%	9·5%	0·5%
As.	66·6	8·5	6·2	22·3%	19·3%	1·3%
Col.	137·8	22·8	8·9	38·8%	46·3%	1·2%
Ban.†	‡	49·9	10·7	34·8%	34·7%	1·1%

* In same age and racial group.
† Figures for Bantu deaths are compiled from certain selected Municipalities.
‡ Unknown because of incomplete birth registration.
Eur. = European; As. = Asiatic; Col. = Coloured; Ban. = Bantu.

Death returns, from which these statistics are compiled, do not reflect the position regarding malnutrition, because only the primary cause of death is used for statistical purposes. Malnutrition, apart from kwashiorkor, is usually listed as a

TABLE III. DEATH RATES FROM ALL CAUSES FOR CERTAIN AGES BY RACE AND PERCENTAGE DUE TO GASTRO-ENTERITIS ALONE: CAPE TOWN, 1958.[4]

Race	Deaths from all causes		Deaths from gastro-enteritis as % of deaths from all causes*	
	per 1,000 live births	per 1,000 population*		
	0 - 1 yr.	1 - 4 yrs.	0 - 1 yr.	1 - 4 yrs.
European	23·3	0·9	1·2%	5·9%
Coloured	85·69	10	35·4%	40·7%
Bantu	191·1†	—	56·1%	39·3%

* In same age and racial group.
† Figures for Bantu must be taken with reserve due to incomplete registration of births.

contributory rather than primary cause of death. It is noteworthy, however, that malnutrition is given as a primary cause of death in Pretoria in 10·7% of non-European deaths between 1 and 5 years of age[2] and in Cape Town in 13% of Bantu deaths between 1 and 2 years.[4]

The total incidence of gastro-enteritis is not known; however, clinics and out-patient departments are inundated with cases of the disease in the summer months. At one hospital in the Cape Peninsula* approximately 9,000 cases of gastro-enteritis were treated in the 6-month period November 1958 to April 1959. Of these, 1,185 needed resuscitation with parenteral fluids.[5]

The extent of the problem presented by mortality from gastro-enteritis is illustrated in Figs. 1 - 5, drawn from Cape Town statistics. These are probably representative of other centres in the Union.

Figs. 1, 3 and 4 illustrate the fact that gastro-enteritis as a cause of death in the whole population is second only to heart disease and overshadows all infectious diseases except tuberculosis, which is also related to malnutrition. Gastro-enteritis exceeds all other causes of death in non-Europeans in the first 5 years of life.

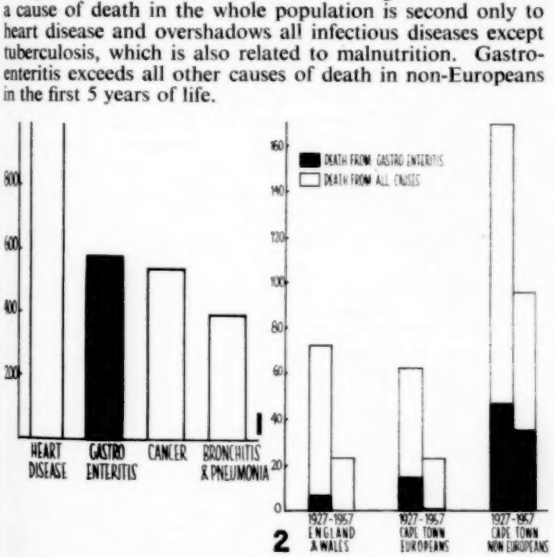


Fig. 1. Number of deaths, all races, from the four principal causes: Cape Town, 1957.[2]
Fig. 2. Reduction in infantile mortality rate (deaths under 1 year per 1,000 live births) in 30 years 1927 - 57, showing rates from all causes and from gastro-enteritis.[2,6]

* See article by Dr. M. D. Bowie at page 344 of this issue.

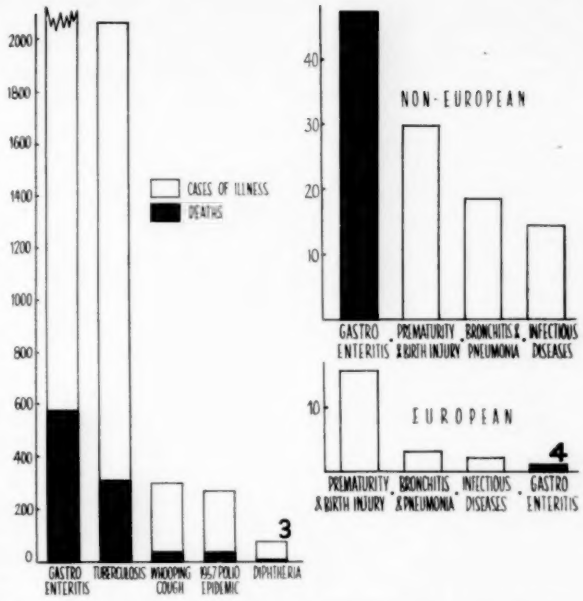


Fig. 3. Number of cases of illness notified and deaths certified from certain infectious diseases: Cape Town, compared with gastro-enteritis: Cape Town, 1957.[2]
Fig. 4. Deaths of children under 5 years from four main causes per 1,000 children born: Cape Town, 1957.[2]

The cumulative number of deaths from gastro-enteritis increases rapidly from 3 months to 2 years, after which age relatively few deaths from this cause occur (Fig. 5). Among non-Europeans these deaths have a markedly adverse effect on the general infantile mortality rate, while among Europeans so few deaths from gastro-enteritis occur that no such effect is seen.

The reduction in the rate of infantile mortality from all causes and from gastro-enteritis in the past 30 years for England and Wales[6] and Cape Town is shown in Fig. 2. Deaths from gastro-enteritis in England and Wales now number about 300 per annum. A similar reduction in infant deaths has occurred in the European population of Cape Town, but in non-Europeans it will be noted that while the infantile mortality rate has dropped appreciably the rate for gastro-enteritis has not dropped proportionately.

FACTORS UNDERLYING HIGH INCIDENCE

1. *Infection*

While there are many non-specific causes of diarrhoea there is little doubt regarding the importance of enteral infections in the aetiology of gastro-enteritis of infancy and childhood. In South Africa salmonella and shigella infections are common. During the summer months in one urban area the incidence of these infections amongst severe cases of gastro-enteritis was found to be as high as 37·5%.[7] It is quite possible that the actual incidence may be even higher than this. The role of specific *Escherichia coli* infection in the epidemiology of gastro-enteritis is as yet uncertain. These organisms were isolated from 41% of cases in one summer outbreak at Pretoria but were also found in 20% of non-diarrhoeal controls.[8] *Bacillus proteus* has also been

found to be present in a large number of cases,[7] but here again its causative role is uncertain. Evidence that virus infections cause outbreaks of gastro-enteritis is at least suggestive,[9-11] and these may be of considerable importance in this country. For example poliomyelitis and Coxsackie virus and antibodies have been found in a high proportion of Bantu infants,[12,13] and it is not unlikely that similar evidence of high infection rates by viruses will be found when appropriate laboratory facilities become more generally available. Intestinal parasites, especially amoebae and *Giardia lamblia*, are frequently found to be responsible for sporadic cases.

Spread of enteral infection is closely related to standards of hygiene and sanitation. In many rural and peri-urban areas safe water supplies and proper sanitation are almost non-existent. The same may be said for fly control.

2. *Malnutrition*

Sixty years ago, in 1900, the Acting Medical Officer of Health for Cape Town, Dr. Averell Alston,[14] reported as follows: 'There is no doubt that the large death rate in regard to the three diseases just mentioned (diarrhoea, thrush and marasmus) is chiefly due to faulty artificial feeding, either by the use of contaminated cow's milk, or farinaceous and other indigestible foods, whereby the intestines are irritated and rendered more liable to septic influences. In regard to the latter, your Council have authorized the appointment of a female inspector, to visit the homes of newly-born children and to advise where necessary. It is essentially a matter of education among the poorer classes of the community, and only by patient perseverance in this respect can we overcome the reluctance of the mothers to discontinue feeding their infants on "anything that's going".'

The appointment of this lady inspector, whose main purpose was the education of mothers in the proper feeding of their infants and the prevention thereby of gastro-enteritis and marasmus, can be considered as the beginning of what today is the Child Welfare Branch of the City Health Department. This service has expanded over the intervening years, to become an integral and important part of the City's health services, with child welfare clinics scattered throughout the Peninsula and the health visitors from the clinics visiting the homes in their areas. The struggle to maintain infant nutrition and to correct malnutrition continues to be one of its major problems. Mothers attending the clinics are supplied with dried milk at cost price, or less where this is needed, and a reasonable standard of nutrition is thereby maintained even in the poorest areas. Although 29,000 individual infants and pre-school children attend the clinics each year, some of these attend only sporadically, and there are many other individuals who do not even take advantage of this service. Many reasons are responsible for this non-attendance, among which are the following: distance from the nearest clinic, economic circumstances which force the mother to go to work, and in many cases apathy or ignorance on her part. It is among the last group of irregular attenders and non-attenders that the worst cases of malnutrition and the greatest number of cases of severe gastro-enteritis are found. In an investigation of 250 deaths from gastro-enteritis[15]

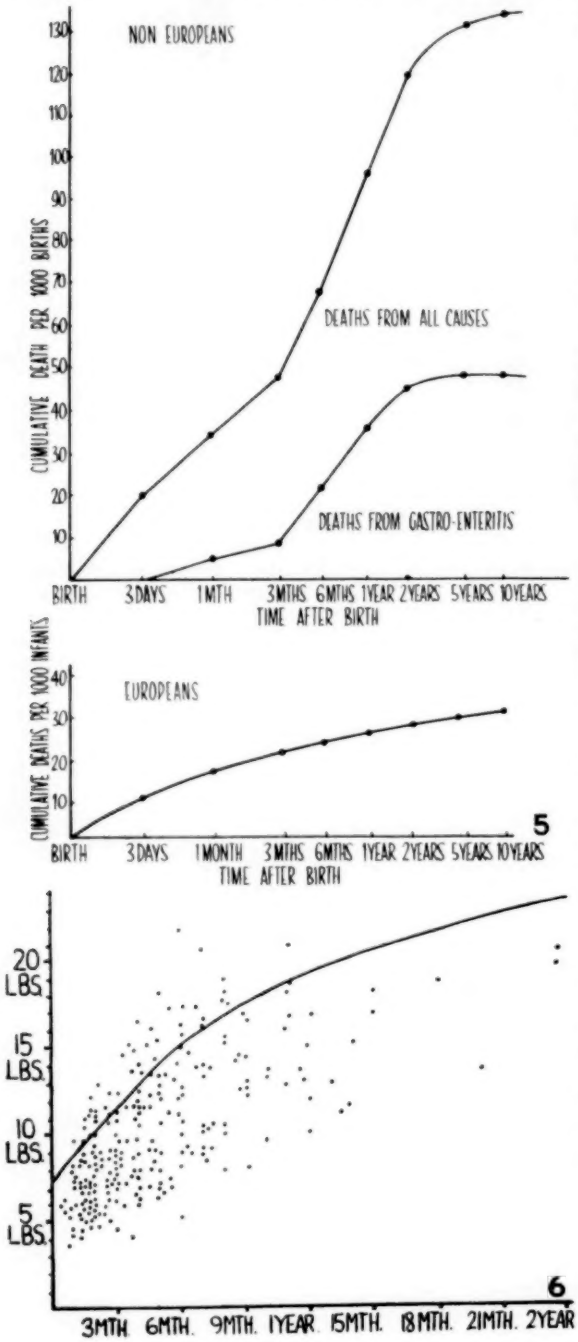
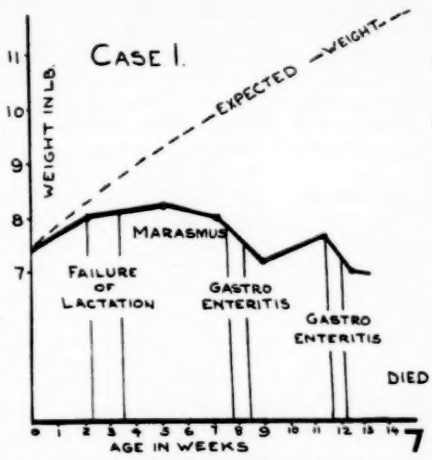


Fig. 5. Cumulative numbers of deaths from all causes and from gastro-enteritis alone, per 1,000 children born; Cape Town. This diagram shows the rapid rise in total deaths between 3 months and 2 years due to gastro-enteritis among non-European infants.[2]

Fig. 6. Weights of children who subsequently died of gastro-enteritis, recorded within a month of death but before the onset of diarrhoea, compared with the average weight of Coloured children. Cape Town.[23]

it was found that 54% of the children who died had never attended a child welfare clinic and 30% had only attended occasionally. In a current study of hospitalized cases of kwashiorkor it was found that 96% of the cases had either never attended a clinic or had done so only occasionally.[16]

In 1907 Finkelstein[17] pointed out the association of malnutrition and summer diarrhoea. In this country, Kahn[7] confirmed the liability of malnourished and marasmic children to develop diarrhoea. This has recently* again been shown in the resuscitation room of the Red Cross War Memorial Children's Hospital at Cape Town.[5] The majority of the children needing resuscitation were malnourished, and the deaths occurred among the most malnourished individuals. In a series of 300 deaths in Cape Town in which the previous history was known, the weights of the children within a month of death but before the onset of diarrhoea are charted (Fig. 6). The chart demonstrates that the majority were severely malnourished before developing gastro-enteritis.



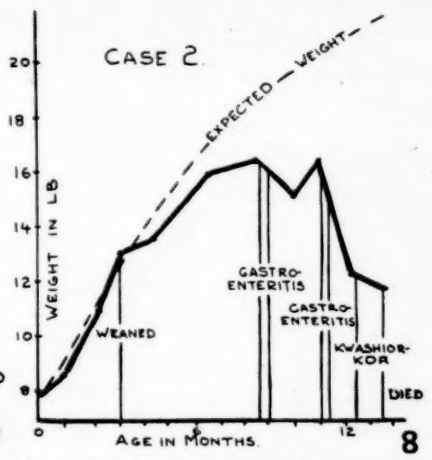


Fig. 7. Diagramatic representation of case history of an infant who died of gastro-enteritis following marasmus.
Fig. 8. Diagramatic representation of case history of a child who died of kwashiorkor following repeated attacks of gastro-enteritis.

While accepting that malnutrition is the forerunner of gastro-enteritis in a high proportion of cases, one must recognize that gastro-enteritis in its turn increases the degree of malnutrition, particularly when milk is withheld for days or weeks after the onset of the attack. A vicious circle is thus established of progressive malnutrition and repeated attacks of gastro-enteritis, which may lead on to kwashiorkor or to a fatal outcome unless proper feeding and treatment are initiated. Two typical case histories (Figs. 7 and 8) are given to illustrate this.

Case 1 shows the downward progress of an infant who started with marasmus after failure of lactation, developed gastro-enteritis, became more marasmic, fell below its birth weight at the age of 9 weeks, and died from a second attack of gastro-enteritis.

Case 2 shows the history of an infant, who progressed normally for 3 months while breast fed, but after weaning failed to maintain his expected gain in weight. He developed gastro-enteritis at 9 months, which accentuated the degree of malnutrition. A second attack of gastro-enteritis ensued, during which he lost weight severely, and just after his first birthday developed kwashiorkor, which proved fatal.

MALNUTRITION IN THE INFANT POPULATION

No figures can be given for the incidence of malnutrition, but it is becoming increasingly apparent that it is high. A recent analysis of weights of all non-European out-patients attending the Red Cross War Memorial Children's Hospital, Cape Town, has shown that half the cases are under the expected weight for age. Of both in- and out-patients 16% are suffering from the grossest degree of both marasmus and kwashiorkor. A current study of a series of hospitalized cases of kwashiorkor and their siblings and near neighbours reveals the existence of a large child population at risk from kwashiorkor.[16] Some children in this population develop the full-blown syndrome; others reach a pre-kwashiorkor state or are saved by timely care or some fortuitous circumstance such as the father's re-employment; the majority survive a childhood of marginal malnutrition with poor growth as the most obvious stigma. The borderline between the sick and the well is a vague one, judging from medico-social data collected. In this susceptible group of children, those that develop the full syndrome of kwashiorkor are the ones subjected to cumulative stresses—environmental, physiological and emotional—which are too great for that individual to deal with at that time. Judging from reports from other parts of the country,[18,19] and from a recent historical review of kwashiorkor,[20] the existence of large child populations at risk is the rule in this country and in many other parts of the world. This population at risk lives on a defective diet from the early months of life. Indeed, one might say that the process starts in intra-uterine life,[21] if a high death rate from still-births and prematurity as seen in non-Europeans in Cape Town (Table IV) are indications of maternal malnutrition.

TABLE IV. STILLBIRTH RATE AND INFANTILE MORTALITY RATE FROM IMMATURITY FOR RACIAL GROUPS: IN CAPE TOWN, 1958[4]

	Eur.	*Col.*	*Afr.*	*As.*
Stillbirth rate	14·2	28·3	52·5	36·4
Infantile mortality rate from immaturity	6·53	16·26	21·15	13·24

Eur. = European; Col. = Coloured; Afr. = African; As. = Asiatic.

Discontinuance of Breast Feeding

In infancy one of the primary factors leading to malnutrition and gastro-enteritis in this socio-economic group is the short duration of breast feeding which the infants are afforded in many instances. In a series of normal non-European infants attending child welfare clinics, it was found that 88% were feeding at the breast at 3 months, whereas in the kwashiorkor series only 47% were still breast

* See page 344 of this issue.

fed at this age. The reason for weaning in 61% of these cases was alleged or actual failure of lactation. Other factors were ignorance, mismanagement, and the mother's return to work. An investigation showed that in the whole Coloured population of Cape Town 39% of the mothers of young infants return to work soon after the infant's birth—17% in industry and 21% in part-time or full-time domestic service.

When artificial feeding is instituted, difficulties arise because of the expense of milk, and because many of the mothers have neither the knowledge nor the facilities to prepare the feeds hygienically and in the right quantities. Once breast feeding is discontinued the following factors are seen to promote malnutrition:

1. *Lack of Hygiene*

This results in frequent infections, particularly of the respiratory and digestive systems, with consequent dietetic difficulties, which tend to produce a state of marasmus.

2. *Incorrect Feeding*

While it is generally accepted by mothers that milk in some form should be given if breast feeding fails, the following practices are common and lead to a greater or lesser degree of malnutrition. They are dictated partly by the relatively high price of milk, and partly by custom and ignorance of food values:

(*a*) *Over-dilution of feeds.* From our experience and that of others in this country[22] this is a very important factor. The mother may mix 1 - 3 teaspoons of dried or condensed milk in a cup of water and feed the baby irregularly on this mixture.

(*b*) *Intermittent milk feeds with substitutes between-times.* The baby may be given 3 milk feeds in the 24 hours, or have milk for only 4 or 5 days in the week and then no more until pay-day. Sometimes the baby may go without milk for a week or longer because money is short. The following are amongst the substitutes commonly given in place of milk feeds: Bottles of plain or sugar water; strained oats, meelbol, Marie biscuits, custard powder, cornflour or other cereals mixed with water or very dilute condensed milk; weak tea, either black or with a little milk. Vegetable pulp, principally squash, is given early by spoon and also replaces milk feeds.

(*c*) *Protein-deficient toddler diet.* From 9 months onwards, milk ceases to be regarded as a necessary article of diet in many families, and the child continues on a diet consisting mainly of cereals and vegetables, especially the popular squash, together with tea or cool drinks. Meat is not given to young children because it is too 'strong'. Fish is the salvation of many, but is usually only available once or twice a week. An important factor in the toddler stage is lack of appetite resulting from various causes such as anaemia and dental caries. As the children grow older they are more demanding and better able to fend for themselves, and are given a more adult diet including meat. Perhaps that is why severe malnutrition and gastro-enteritis are less common after 3 years of age. There is no doubt, however, judging by growth curves,[23,24] that suboptimal nutrition persists throughout childhood.

3. *Environmental and Social Factors*

(*a*) *Housing standard.* The death rate from gastro-enteritis varies markedly in different areas according to the degree of poverty and bad housing present. In two purely non-European areas in Cape Town, namely Athlone and Windermere, this difference is clearly seen. In Athlone, where housing conditions are fairly satisfactory, with one-third of the population resident in municipal houses and many of the remainder reasonably well housed, the death rate from gastro-enteritis is half what it is in the Windermere area, where housing and environmental hygiene are of an extremely low standard. In a previous article[15] it was shown that only 9% of families where deaths from gastro-enteritis had taken place were decently housed. In Krige's study of the social background of malnutrition and gastro-enteritis at King Edward VIII Hospital in Durban[18] it was found that in the urban cases 93% came from shack areas and backyards.

(*b*) *Ignorance.* Lack of proper care resulting largely from ignorance must be indicted as an important causative factor. This includes the general ignorance of child care that prevails amongst the mothers themselves and among the busy neighbours and irresponsible young girls who are often left in charge of infants during the day. The harm that arises from foster care in early infancy is seen most markedly in infants whose mothers return to domestic service and place them with foster mothers. This is often done within the first month. The death rate among these foster-infants is found to be twice that among the Coloured infant population as a whole. There is also the ignorance that results from a poor standard of education among parents, which limits their ability to buy or budget wisely, to reject superstitious practices, and to discriminate between sound advice and plausible sales talk. Many workers, but notably Spence and his group,[25] have concluded that the determining factor in the well-being of the child is the capacity of the mother.

(*c*) *General social disorganization.* Following Krige's classification[18] this term covers a variety of factors that are repeatedly found to play a significant part in the background of malnourished children; viz. unemployment or casual employment of fathers, absence of mothers, illegitimacy, negligence, non-support, alcoholism, and disrupted home and family life. These are especially noticeable in urban areas where rapid industrial development, causing among other things a shift in population from country to town, is producing a demoralized and insecure society that has not yet succeeded in adapting itself to the pace of current change.

(*d*) *Poverty.* Underlying all the social causes outlined above is the fact that there is widespread poverty among the non-Europeans in the Union, and that a considerable section of the population do not earn enough to cover their basic needs. This is illustrated in Cape Town by the following figures. In 1955, Prof. Edward Batson conducted a housing survey of Cape Town from the findings of which it appeared that 5% of all Coloured households in the municipality had gross monthly incomes of less than £10, and 25% had gross monthly incomes of less than £20. The corresponding percentages among European households were 1 and 4 respectively.[26] In a current survey of 300 Coloured families in Cape Town in which deaths from gastro-enteritis had occurred, it was found that 80% had a total income of under £20 a month, and in 30% the wage earner was unemployed or in casual employment and earning less than £10 a month.

HOW CAN THE POSITION BE IMPROVED?

The following are means by which improvement can be effected in the present position:

1. *Promotive Health Services*

Child welfare services embracing efficient home visiting of infants and pre-school children, and infant welfare clinics, are extremely important in the maintenance of the nutrition and health of the pre-school child. This is a pressing need for extension of these services, to cover all infants and pre-school children. Through this medium the following health measures can be achieved:

(*a*) Promotion of breast feeding, which is the surest safeguard against malnutrition and gastro-enteritis in infancy. This can best be encouraged by sympathetic propaganda among mothers at pre- and post-natal clinics, combined with measures to ensure proper nutrition of the mothers. In order to obviate early weaning, mothers should be encouraged to keep their infants in their care and not to return to work and leave them in the care of incompetent persons. To this end it would be of great assistance if the period of maternity benefit for women employed in industry could be extended beyond the present limit of 8 weeks after the infant's birth, and if domestic servants were allowed to have their young infants with them when returning to work.

(*b*) The provision of subsidized milk supplies for bottle-fed babies as well as for expectant and nursing mothers. This provision is necessary on an extensive scale if infant nutrition is to be maintained at a reasonable level.

(*c*) The distribution of milk, either as liquid milk or dried skimmed milk of high quality, and other protein-containing foods such as pulses or wholemeal bread and peanut butter, to the large number of pre-school children who are at present suffering from deficiency of protein in their diet.

(*d*) Improvement in the supervision of children between the ages of 1 and 6 years. This service is very necessary, for on the whole this group receives the least attention from health officials, falling between baby clinics on the one hand, and school inspections on the other.

(*e*) Education of mothers in the principles of hygiene and nutrition. This can be done by home visists and talks by health visitors or health educators, and by posters, pamphlets, lecture-demonstrations and films.

(*f*) Teaching of family spacing, to reduce the sizes of families to reasonable numbers, so that the parents are better able to care for their children.

All this increase of clinic services will need greater subsidies from local and national authorities, particularly for the provision of milk and other food supplements. It may be pointed out that machinery exists in the Public Health Act No. 36 of 1919 to deal with this problem. Section 3 of the Act includes among the functions of the Department of Public Health 'the prevention . . . of . . . preventable diseases within the Union.' To this end the National Nutrition Council was established by the Public Health Amendment Act No. 14 of 1940. The function of this Council 'shall be to investigate and report . . . upon all matters relating . . . to the prevention of malnutrition in and the improvement of the diet of the inhabitants of the Union . . .'

2. *Curative Services*

At present some of the larger hospitals and dispensaries in the Union have special resuscitation rooms in their out-patient or casualty departments which are properly equipped for the administration of intravenous or subcutaneous fluids.[27,28,5] At these centres large numbers of children suffering from gastro-enteritis are successfully treated as out-patients.

Equipment of this type of resuscitation should be extended to more hospitals and out-patient dispensaries near to affected population groups. The cost of running a service of this nature is relatively small when the number of lives saved is taken into consideration. All such departments should be orientated towards education of the mothers in nutrition. In this connection it is important that children with gastro-enteritis should be given milk feeds within 24 hours of commencing treatment. Many children become severely malnourished because milk is withheld for too long a period after an attack of gastro-enteritis.

Convalescent care. Follow-up care at home, and the provision of convalescent beds for young children, are both badly needed to ensure proper recovery and restoration of nutritional state after gastro-enteritis, marasmus, and kwashiorkor. Lack of such care frequently undoes the good achieved by hospital treatment and is responsible for much of the progressive malnutrition and repeated attacks of gastro-enteritis which so frequently occur. Closer co-ordination of curative services on the one hand and child welfare services on the other is essential. This is being effected with great success in the Cape Peninsula, where cases seen at Provincial hospitals are followed up by the staff of the local child welfare clinics. Added to this is the close liaison and consultation between the professional staff of the Cape Town muncipal child welfare department and their *vis-à-vis* in the Provincial service.

3. *Social Provisions*

Improvements in the current wage structure of unskilled workers and the progressive abolition of poverty must undoubtedly reduce the incidence of malnutrition and gastro-enteritis. This has already been observed in Britain over the last 10 years, where improved social conditions have been reflected by a marked improvement in child health. In 1947 the infantile mortality rate per 1,000 live births from all causes was 41, and from gastro-enteritis alone 5·13, whereas in 1957 the infantile mortality rate from all causes had dropped to 23 and that from gastro-enteritis alone to 0·44. Before 1947 the rates had been fairly stationary for some years.[6]

Short of such fundamental changes in the country's economy, much can be done by provisions such as the following:

(*a*) *Assistance for children of working mothers.* More crèches are needed for the children of working mothers. These can be run by voluntary bodies, by local authorities, or as an adjunct to factories where women are employed.

(*b*) *Insistence on parental responsibility.* The Children's Act, at present in the course of being amended, should in its new form be so drafted as to deal more effectively with neglect and failure on the part of the father to supply proper maintenance, especially where much of the available money is squandered on alcohol.

(*c*) *Recruitment of public interest and support.* All employers of labour, whether domestic, manual or industrial, could educationally and materially assist in one way or another (as previously mentioned) by encouraging their workers to provide proper care for their children and thereby improve the general health of the rising generation.

4. *Non-personal Health Services*

From the improving of housing, water supplies and sanitation, and the controlling of fly breeding, a reduction in the spread of the infectious forms of gastro-enteritis will result; but where malnutrition preceeds or predisposes to gastro-enteritis the nutritional provisions outlined above must be applied so as to prevent what today is a preventable disease which is exacting an intolerable price in the loss of young lives.

SUMMARY

In this paper we have attempted to show in some detail the extent of the problem of gastro-enteritis in pre-school children and infants, particularly amongst the Coloured and Bantu. The relationship of malnutrition to gastro-enteritis has been shown. It is in the main the child that is already suffering from malnutrition who becomes a victim, and a vicious circle is frequently set up, worse malnutrition following each repeated attack of gastro-enteritis, leading finally to kwashiorkor or death. The social and nutritional factors responsible for this state of affairs are discussed, and the preventive, curative and social measures suggested whereby this preventable disease and its sequelae can be markedly reduced.

We wish to thank Dr. E. D. Cooper, Medical Officer of Health for Cape Town, and Prof. Findlay J. Ford, Professor of Child Health, University of Cape Town, for their assistance in the preparation of this paper, and for permission to publish certain information.

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THE MANAGEMENT OF GASTRO-ENTERITIS WITH DEHYDRATION IN OUT-PATIENTS*

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Gastro-enteritis in non-European infants remains a serious public-health and paediatric problem in South Africa. In the major cities of the Union over 10,000 children die every year from this disease.[1] Of every 1,000 non-European children born, 135 do not reach school age, and 49 of these die of gastro-enteritis between the ages of 3 months and 2 years. In comparison, of every 1,000 European children born, only 29 fail to reach school age, and of these 1 may die from gastro-enteritis.[1] In Cape Town over the past 20 years there has been a steady decline in the infantile mortality rate in both non-Europeans and Europeans. In European infants there has been a concomitant fall in the mortality rate from gastro-enteritis, until in 1957 it was 1·4 per 1,000 live births, but in non-European infants there has actually been a slight rise in the infantile mortality rate from this condition[2] (Fig. 1).

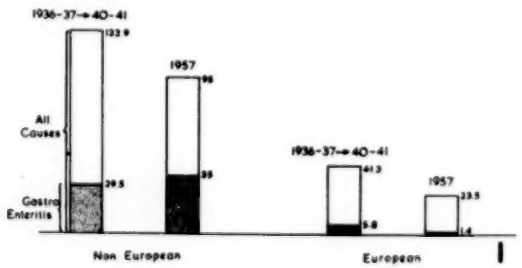

Fig. 1. Annual infant mortality rates, City of Cape Town for the 5 years ended 30 June 1941 and for the year 1957.

Dehydration and metabolic imbalances are potent factors in the causation of death in these infants. In-patient hospital care is possible for only a very small minority, for the hospital beds available are totally inadequate to deal with the overwhelming numbers that present for treatment. The majority have to be managed as out-patients. Resuscitation by means of parenteral fluids given in the out-patient department is used in several centres.[3,4] This paper describes the method used at the Red Cross War Memorial Children's Hospital and reviews the findings and results over a period of 6 months.

Cases in the series. During the 6-month period November 1958 to April 1959 (see Table I) an estimated 9,000 cases of gastro-enteritis were seen in the out-patient department. Of these, a total of 1,185 children were dehydrated and received parenteral fluid therapy in the out-patient resuscitation room. Clinical data in this report were obtained by analysis of the 1,185 children presenting with dehydration. Of these children 161 attended only once or failed to attend until complete recovery, and are not considered in the assessment of results.

* Paper presented at the 42nd South African Medical Congress (M.A.S.A.), East London, C.P., September - October 1959.

TABLE I. GASTRO-ENTERITIS: 6 MONTHS, NOVEMBER 1958 - APRIL 1959

Estimated number of cases seen	9,000	
Total number of cases with dehydration ..	1,185	
Number of cases outcome unknown	161	(13·6%)
Total number of cases included in series ..	1,024	
Number of cases admitted	101	(9·9%)
Number of cases treated solely in out-paitient department	923	

Of the remaining 1,024 cases, 101 children were admitted either because of the failure of out-patient treatment or because of the occurrence of complications. In easier circumstances many more would have been admitted at their first attendance.

Seasonal incidence. Gastro-enteritis occurs throughout the year but, as is well-known, there is a marked seasonal increase during the summer months. This is reflected by the

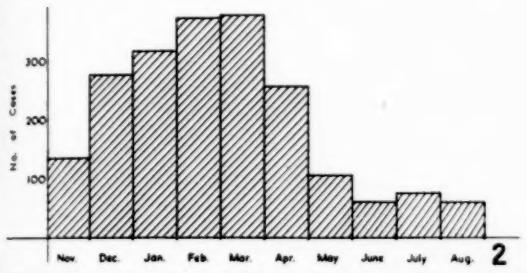

Fig. 2. Gastro-enteritis with dehydration: seasonal evidence of cases.

number of cases receiving parenteral fluids every month (Fig. 2). As can be seen, the largest number of cases occurs between December and April, with a peak incidence during February and March. These findings parallel the seasonal occurrence of deaths from gastro-enteritis in Cape Town.[5]

Race and sex. Of the 1,185 children treated, 806 were Coloured and 379 were African. There were slightly more males than females in each group, giving a total of 651 males and 534 females.

Age incidence. Analysis of the ages of the infants treated with parenteral fluids (Fig. 3) shows that the incidence of gastro-enteritis with dehydration is highest at the ages of 2-6 months, with the peak incidence occurring during the 3rd and 4th months of life. After the age of 6 months the incidence drops sharply, but a significantly large number of cases were seen and treated up to the age of 2 years. After

Fig. 3. Gastro-enteritis with dehydration: distribution of cases and deaths by age (in months.)

2 years only sporadic cases of gastro-enteritis with dehydration occurred. The age incidence seen in this series again parallels that found in a study of deaths from gastro-enteritis in Cape Town[5] and is similar to the age incidence found elsewhere by various authors.[2,3,6,7] The high incidence of gastro-enteritis and dehydration in the younger infant with its relatively greater water content and turn-over emphasizes the fact that it is the younger infant that requires parenteral fluid therapy most frequently.

Nutrition. Malnutrition predisposes to illness of all kinds and gastro-enteritis is no exception. In the well-nourished European population gastro-enteritis is a mild disease with a low mortality. In the non-European population, where malnutrition is rife, gastro-enteritis is a major killer. In this series (Fig. 4) if the average weight found for each month of life is taken it is only during the first month that the average weight approaches that of the third percentile of the Anthropometric Chart of the Children's Medical Centre, Boston. This discrepancy in weight increases with increasing age, and at 1 year the average weight of the infants is only 13 lb. 8 oz. as compared with the lowest American figure of 18 lb. 8 oz. This difference cannot be accounted for by dehydration. In severely dehydrated infants the gain in weight after rehydration may be up to 17% of the original weight, but on an average it is 10%.[4] If the average weight per month of life of those children who died is studied, it is found that their weights are below those of the series in general and that this discrepancy also increases with increasing age. At 1 year the average weight of the children dying is only 12 lb. Malnourished children have a relatively greater proportion of water to total body mass than adequately nourished children,[8] and so loss of water has serious consequences in malnourished infants.

TREATMENT

A standard regime is used at this hospital in the treatment of gastro-enteritis.[9] In children presenting without clinical evidence of dehydration, milk feeds are stopped for 24 hours and the infant put on half-strength Darrow's solution in 2½% dextrose water by mouth. The mother is advised to give the infant small but frequent feeds of this solution with a total intake over the first 24 hours of 3-4 oz. per lb. body-weight. Thereafter the infant is put on to milk feeds. These are also given in amounts to make a total intake of 3 oz. (or slightly more) per lb. body-weight per day. The milk feeds are given as full-strength milk, and no attempt is made to start with diluted feeds and work up to full strength gradually. Skimmed milk is usually given because it is cheap and readily available. Occasionally, after 24 hours on half-strength Darrow's solution, the infant is put back onto full-strength full-cream milk feeds. Our impression is that the type of milk feed makes very little difference. The initial 24 hours on half-strength Darrow's solution seems, however, to be of importance. Infants are not kept on Darrow's solution for longer than 24 hours unless vomiting persists. To keep a child on clear fluids orally until diarrhoea has ceased is unnecessary and only adds to the usually coexistent malnutrition.

Routine antibiotic or chemotherapeutic agents are given to all children with gastro-enteritis. The majority initially receive one dose of long-acting penicillin by injection and a soluble sulphonamide orally for 5 days. If there is no response

to treatment, or if deterioration in the infant's condition occurs, therapy is usually changed to oral chloromycetin. Most patients respond to penicillin and sulphadiazine. Occasionally, when an infant presents with a history of blood in the stools chloromycetin is used from the commencement of treatment.

Parenteral Fluid Therapy

Indications. Parenteral fluid therapy is indicated if there is more than minimal dehydration, acidosis, shock, or intractable vomiting. Signs of dehydration include loss of tissue turgor, sunken dry eyes, sunken fontanelle, and a dry mouth and tongue.

Parenteral fluids are given either subcutaneously or intravenously. In this series, 480 infants were initially given subcutaneous fluids and 705 intravenous therapy. Subcutaneous fluids were administered to infants with mild dehydration and who were not shocked or acidotic. The amount of fluid to be given is calculated on the estimated degree of dehydration and the body weight. Practically speaking the subcutaneous fluids are usually given in amounts approaching 10% of the body weight. The total amount is given either as half-strength Darrow's solution in 2½% dextrose water, or half the quantity is given as half-normal saline in 2½% dextrose water and the other half as Darrow's solution. The fluids are administered into two sites, usually the lower part of the axillae. Hyaluronidase is given to promoted spread and absorption.

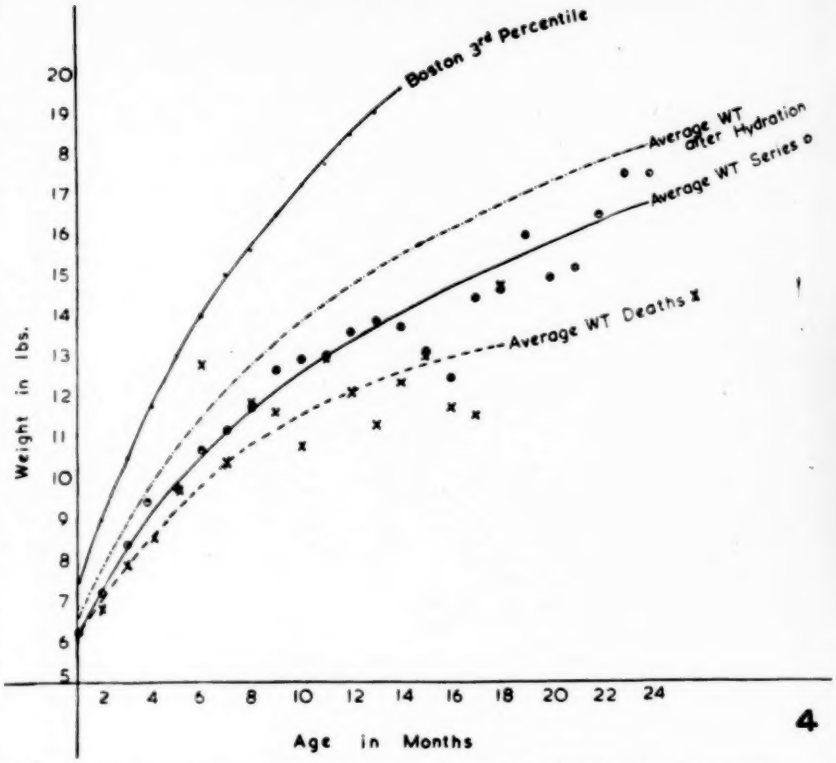

Fig. 4. Gastro-enteritis with dehydration: average weight by age (in months) of cases, cases after hydration and deaths. Boston 3rd percentile curve shown for comparison.

Intravenous fluids are administered to all severely dehydrated infants or those presenting with dehydration and signs of circulatory collapse or acidosis. Most of the infants receiving intravenous therapy are kept on the drip from 12 to 24 hours whilst the replacement and maintenance fluid for that period is given. Both replacement and maintenance fluid are given as half-strength Darrow's solution in 2½% dextrose water. Small oral feeds of the same solution are also given. As with subcutaneous fluids, the amount given is calculated according to the degree of dehydration and the body weight. In practice the degree of dehydration is usually taken as 10%, and so in 24 hours the infant gets 50 c.c./lb. body-weight as replacement and 80 c.c./lb. as maintenance fluid, e.g. a total of 130 c.c./lb./day. Intravenous fluids are administered in scalp veins or occasionally, in older children, in arm veins. It has very seldom been found necessary to do a 'cut down' on a vein to administer intravenous fluids.

The resuscitation room. This is a large room in the out-patient department fitted with several examination couches with overhead beams attached from which intravenous drip sets may be hung (Fig. 5). All non-European cases of gastro-enteritis with dehydration presenting at the hospital are initially treated in this room. The mother or responsible relative stays with the child while parenteral fluids are being administered and takes the infant home when it is rehydrated. The infant is seen the following day and progress checked; if indicated, further parenteral fluids are given. Only a small percentage (9·8%) eventually have to be admitted to the wards for in-patient treatment.

Results of Treatment

Most of the cases recovered within 3 or 4 days of rehydration, and required no further treatment. In 17% of the cases repeated parenteral fluid infusions were required for correction of recurrent dehydration. Of the 1,024 cases on which we had adequate follow-up record, 97 died. Of these children, 68 died at home or in the out-patient department. Very few died while actually receiving parenteral fluids. There were also 29 who died after admission to the wards. This gives a total mortality rate of 9·5% for *all cases of gastro-enteritis with dehydration* treated during the 6 months under review. In the 923 cases treated solely as out-patients, there were 68 deaths, giving a mortality rate of 7·4%. In the 101

Fig. 5. Resuscitation room for out-patients at the Red Cross War Memorial Children's Hospital, showing couches with overhead beams for intravenous drip sets.

patients admitted there were 29 deaths giving a mortality of 28·7% (Table II).

TABLE II. MORTALITY

		% *Mortality*
Total number with known outcome ..	1,024	
Total number of deaths	97	9·5%
Number treated solely in out-patient department	923	
Number of these who died	68	7·4%
Number of cases admitted	101	
Number who died after admission	29	28·7%

It is difficult to find mortality figures to compare with our findings. Practically all published figures relate to gastro-enteritis with or without dehydration and not as in our series only to those with dehydration. Before parenteral fluid therapy was introduced shortly before 1930, the death rate in infants suffering from gastro-enteritis was about 50%.[6] After its introduction the mortality rate was rapidly halved and there has been a steady decline in mortality with the introduction of antibiotics and isolation techniques. The mortality in Toronto is now less than 1% and other series from Western European countries show an equally low death rate. In the trials of the Medical Research Council on gastro-enteritis (1953), the mortality rate was 3%.[10] Among European in-patients at the Red Cross War Memorial Children's Hospital in 1958 there were no deaths.

Non-European in-patient mortality figures reported from other centres vary from 25%[11] to 44%[12] and are comparable with the mortality in in-patients in this series. The children that are admitted are the most severely ill, and often have contributing parenteral illness or complications of water and electrolyte imbalance. The mortality rate for out-patients (7·4%) compares favourably with that obtained in a similar series done at Groote Schuur Hospital,[4] where the mortality was found to be 13%, even though that study included infants without dehydration. In that series the only parenteral fluids given were subcutaneous infusions, and this supports our impression that intravenous fluids are more effective than subcutaneous fluids.

The largest number, approximately 50% of the total number of deaths, occurred in the age group under 5 months. There were no deaths in this series after the age of 18 months, and very few over the age of 1 year. Only 11 deaths occurred in the age group 13 - 18 months. Heavier mortality took place in the age group 10 - 12 months. Malnutrition is a possible, and indeed a highly probable, contributing factor here (Fig. 3).

As can be seen from the figures presented, a large number of children severely ill with gastro-enteritis were treated during the 6 months under consideration. When the social background and state of nutrition of these children is taken into account, the total mortality rate of 9·5% is satisfactory and surprisingly low. As the number of non-European in-patient hospital beds cannot possibly cope with the number of children presenting with gastro-enteritis and dehydration, a resuscitation room in the out-patient department is a vital and indispensable part of the arrangements required to cope with the situation. This type of treatment could also be given at some peripheral clinics and so relieve the hospitals of a considerable part of their very heavy case load. It would not be necessary for specially skilled medical staff to give the treatment, since peripheral clinics would almost certainly deal with the infants at an earlier stage of their illness, when it would be sufficient to inject the fluid subcutaneously. Even the more seriously dehydrated and shocked children would benefit by receiving such treatment while awaiting transport to the bigger centre where, if no other arrangement could be made, out-patient intravenous therapy could be undertaken. Further daily subcutaneous infusions could then be given at the peripheral clinics, if thought necessary. Such an arrangement would clearly be of advantage to all, not least the patient.

SUMMARY

1. The extent of the gastro-enteritis problem in non-European infants at this hospital is indicated—about 9,000 cases presenting in 6 months.

2. The number of these who were found to be suffering from dehydration amounted to 1,185.

3. Only 101 could be admitted; 1,084 had out-patient treatment only.

4. The close relationship between malnutrition and gastro-enteritis is shown. Practically all the patients were malnourished and those children who died showed the grossest degree of malnutrition.

5. Rehydration by the subcutaneous and intravenous routes is discussed.

6. The recovery rate in out-patients is gratifyingly high.

7. The death rate in in-patients was 28·7%, for the series as a whole 9·5%, and for out-patients alone 7·4%.

8. A plea is made for the initiation of peripheral clinics, where much of the rehydration work could be undertaken, with consequent benefit to all concerned.

ADDENDUM

Since the completion of this paper, attempts have been made to trace the 161 cases in which the outcome was unknown. Over 100 of these children have been traced and the mortality rate among them was lower than that for the series as a whole, being about 5%. It transpired that many of these infants had been referred to hospital from peripheral clinics for parenteral fluid therapy. Follow-up was undertaken at the clinic and the child was not sent back to hospital. In other cases the infant had responded to treatment so rapidly that the mother did not consider it necessary to bring the child back.

I wish to thank Dr. J. Mostert, Medical Superintendent of the Red Cross War Memorial Children's Hospital, for permission to publish this paper; Prof. F. J. Ford and Dr. J. D. L. Hansen for their stimulus and advice; Dr. S. R. Esrachowitz for invaluable help; Dr. V. C. Harrison, Mrs. M. Craigen and Miss D. Barker for assistance with the records; and Miss C. Freesemann for the graphs.

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A CASE OF WIDESPREAD URTICARIA PIGMENTOSA WITH EPISODES OF FLUSHING

JEAN WALKER, M.D. (CAPE TOWN), *Durban*

Urticaria pigmentosa was first described by Nettleship in 1869. Since then more than 500 cases have been reported, amongst which flushing was noted in only 19 cases—14 of widespread cutaneous urticaria pigmentosa and 5 of solitary mastocytoma. It is indeed peculiar that so few cases of vasomotor disturbances have been reported when one considers that urticaria pigmentosa is a mast-cell reticulosis[1] and that mast cells are known to liberate powerful vaso-active substances, particularly histamine and 5-hydroxytryptamine.

Marshall[2] has seen 2 further patients with solitary mastocytoma associated with flushing and also a woman with scattered pigmented lesions and vasomotor disturbances so severe that they frighten her children. The case of widespread urticaria pigmentosa with flushing described by Birt and Nickerson[3] and the 2 cases reported by Neill[4] are the most recent to be recorded.

The following case is recorded in view of the extreme rarity of the phenomenon of flushing with any of the mastocytoses:

CASE REPORT

A.W., a European boy aged 16 months, was apparently normal at birth. There was no family history of any skin disease.

When he was 2 weeks old blisters began to appear on his scalp; these dried and formed brown crusts, which fell off leaving light-brown marks. About 2 weeks later, brown spots and patches appeared in large numbers on the rest of the body, blisters only forming on those which were subjected to mild trauma like bumping or rubbing.

At the age of 2 months the first flush appeared, the whole skin except that on the lower legs being suffused with a uniform bright redness which, however, did not distress the baby in any way. Thereafter, flushing occurred at intervals of about 1 month until he was 13 months old, when there were no further episodes until 2 days before I saw him at the age of 16 months. This attack occurred on his waking early in the morning. The flushes lasted about three-quarters of an hour and then rapidly subsided. They seemed to arise quite spontaneously and did not follow trauma to the skin, or urticaria or blistering of the lesions, or the ingestion of food.

When the baby was 5 months old he lay on a cotton reel and a small raised nodule developed at the site of contact on his back. Blisters formed on the nodule from time to time but this did not coincide with the episodes of flushing. The first flush actually occurred 3 months before the nodule appeared on the back.

The normal skin between the pigmented lesions did not exhibit demographism.

The baby was otherwise very healthy and was never in any distress from pruritus during the flushing attacks or during the intervening periods, although he often rubbed one large brown macule in his groin whenever his napkin was removed. He never suffered from dyspnoea or gastro-intestinal upsets.

Examination. The patient was a fine healthy boy. His whole skin from scalp to feet was practically covered with well-defined, discrete, brown macules, irregular in shape and size varying in colour from pale beige on the exposed parts to dark sepia on the trunk. This difference in colour was very striking and looked as though exposure to light might be having some beneficial effect on the pigmentation. Only one raised nodular lesion was seen, the one described as following trauma to the back from a cotton reel. This nodule was oval, about 1 cm. long, and of a peculiar pale lilac colour which did not fade on diascopic pressure; the surface showed *peau d'orange*. Rubbing of this nodule, and indeed of several of the macules, produced immediate local erythema which was followed by whealing and the fairly rapid formation of a bulla, although no general flush was provoked by these measures. The normal intervening skin did not urticate on rubbing.

The *urine* was examined for 5-hydroxy-indole acetic acid and none was found; the specimen was not taken during a flushing episode. Unfortunately, urinary histamine estimations were not possible in the laboratory to which the urine was sent.

Histology. The nodule on the back was excised and histological sections showed numerous spindle-shaped cells in the corium and upper dermis. These cells contained granules which stained metachromatically with toluidine blue. The infiltrate extended right up to the epidermal basal layer. Disruption of the collagen was evident and the tissue infiltrate was associated with a fine reticular structure. There were no significant epidermal changes. The histological picture was consistent with that of mastocytoma.

I wish to thank Dr. W. Mukheiber for his cooperation and interest in this case.

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UNIVERSITY NEWS : UNIVERSITEITSNUUS

UNIVERSITY OF NATAL MEDICAL STUDENTS' COUNCIL : FIRST CLINICAL CONFERENCE

This Conference will be held on 3 - 5 May 1960 in the Upper Lecture Theatre, Medical School, Umbilo Road, Durban. The theme of the Conference will be 'Some problems of the newborn'. Further information may be obtained from Dr. V. K. G. Pillay, King Edward VIII Hospital, Durban.

PROGRAMME

First Session, Tuesday 3 May, 8.15- 10.15 p.m.

Chairman: Dr. H. L. Wallace. Welcome by Dr. V. K. G. Pillay.

'Antenatal paediatrics'—Dr. F. P. Walt.

'Comparative mortality rates'—Mr. D. D. Rangiah.

'Medical emergencies'—Dr. P. Klenerman.

'Surgical emergencies'—Prof. A. E. Kark.

'Fluid balance in the newborn'—Dr. N. M. Mann.

Discussion: Opening speaker—Dr. Alan B. Taylor.

Second Session, Wednesday 4 May, 8.15 - 10.15 p.m.

Chairman: Mr. A. C. Copley.

'Statistics of congenital abnormalities'—Mr. J. R. Domingo.

Discussion panel: 'When to treat'.
'Modern views on the causes of congenital abnormalities'—Prof. T. Gillman.
'The surgical approach to hydrocephalus'—Mr. M. J. Joubert.
'Care of the meningocele'—Mr. D. S. Chapman.
'Circumcision'—Mr. N. R. Butcher.
'Imperforate anus'—Prof. A. E. Kark.
'Erb's palsy, clubbed foot, congenital dislocation of hip'—Mr. J. D. Raftery.
'Cleft palate and hypospadias'—Mr. B. W. Franklin Bishop.
Discussion: Opening speaker—Mr. A. C. Copley.

Third Session, Thursday 5 May, 8.15 - 10.15 p.m.
Chairman: Prof. I. Gordon.
'Hyperbilirubinaemia'—Dr. B. G. Grobbelaar.
'Neonatal infection'—Mr. S. L. V. Zwana.
'Tetanus neonatorum'—Prof. E. B. Adams.
'Resuscitation of the newborn'—Dr. B. G. Jackson.
'What shall we tell the parents?'—Dr. H. L. Wallace.
Discussion: Opening speaker—Dr. W. E. B. Edge.

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

MINUTES OF AN EXTRAORDINARY GENERAL MEETING OF THE MEDICAL ASSOCIATION OF SOUTH AFRICA, HELD AT MEDICAL HOUSE, 35 WALE STREET, CAPE TOWN, ON FRIDAY 26 FEBRUARY 1960, AT 3 P.M.

There were 3 members present. Dr. A. P. Blignault proposed that Dr. T. Shadick Higgins be the Chairman of the Meeting. There were no other nominations and Dr. Shadick Higgins thereupon presided.

The Secretary submitted the notice calling the Meeting which, on the proposal of Dr. Blignault, it was *Agreed* should be taken as read.

The Secretary then submitted the proposed alterations to Articles 23(*b*) and 25 of the Association, as follows:

I. That Article 23(*b*) be and is hereby cancelled and deleted and that the following new Article 23(*b*) be and is hereby substituted therefor:

'23(*b*) The reception of such addresses, and especially the address to the Association of the President, as the Council shall have arranged to be received at such meeting'.

II. That Article 25 be and is hereby cancelled and deleted and that the following new Article 25 be and is hereby substituted therefor:

'25. The Council shall from time to time arrange meetings or conferences, alone or in conjunction with other bodies, which it shall be open to every member of the Association to attend, for the purpose of receiving addresses or other communications relating to the medical or allied sciences, and discussing such subjects pertaining thereto as the Council may think proper, and the Council may arrange to hold such meetings or conferences within or without the Union of South Africa. When held in the Union of South Africa, such meeting shall be convened at the same place as the Annual General Meeting of the Association and in connection but so as not to conflict therewith'.

It was proposed by Dr. Shadick Higgins, seconded by Dr. Blignault and *Resolved*—

'That the present Articles 23(*b*) and 25 be and are hereby cancelled and deleted, and that the following new Articles 23(*b*) and 25 be and are hereby substituted therefor:

"23(*b*) The reception of such addresses, and especially the address to the Association of the President, as the Council shall have arranged to be received at such meeting."

"25. The Council shall from time to time arrange meetings or conferences, alone or in conjunction with other bodies, which it shall be open to every member of the Association to attend, for the purpose of receiving addresses or other communications relating to the medical or allied sciences, and discussing such subjects pertaining thereto as the Council may think proper, and the Council may arrange to hold such meetings or conferences within or without the Union of South Africa. When held in the Union of South Africa, such meeting shall be convened at the same place as the Annual General Meeting of the Association and in connection but so as not to conflict therewith".'

There was no further business and
the Meeting ended at 3.10 p.m.

THE BENEVOLENT FUND : DIE LIEFDADIGHEIDSFONDS

The following donations during January and February 1960 are gratefully acknowledged:

Met dank word die volgende skenkings gedurende die maande Januarie en Februarie 1960 erken:

Votive Cards in Memory of: Geloftekaarte ter Nagedagtenis aan:
Mrs. R. W. Normand *by* Dr. and Mrs. W. Gilbert and Dr. A. W. Sichel; Dr. D. H. R. Vollet *by* Mr. C. Weinberg, Natal Coastal Branch (M.A.S.A.) and Mr. A. J. S. Boyd; Dr. A. S. Parker *by* Natal Coastal Branch (M.A.S.A.), Dr. B. A. Armitage and Dr. R. B. Peckham; Dr. F. H. Molliere *by* Natal Coastal Branch (M.A.S.A.); Mrs. R. Forsdick *by* Dr. Lance Knox; Mrs. English *by* Dr. H. A. Kalley and Dr. R. B. Peckham; Mr. Clarke *by* Dr. B. A. Armitage; Dr. Hugo *by* Dr. A. L. Agranat; Dr. G. W. Doran *by* Border Branch (M.A.S.A.); Mr. A. H. Krynauw *by* Mr. W. G. McDavid and Dr. R. Geerling; Dr. G. W. S. Klooster *by* Dr. R. Geerling; Mr. Jack van Renen *by* Dr. P. A. Smuts; Dr. T. Mulock-Bentley *by* Dr. V. N. Larsen; Dr. L. F. B. Biccard *by* Dr. A. W. Sichel, Dr. G. M. Malan and Mr. R. W. Normand; Dr. T. D. Shanks *by* Dr. P. C. W. Madden and Dr. T. E. Hofmeyr; Dr. McGinn *by* Natal Coastal Branch (M.A.S.A.) and Mr. V. N. Larsen.

Total received from Votive Cards: £34 16s. 6d.
Totaal ontvang van Geloftekaarte:

Services rendered to: Dienste gelewer aan:
Mrs. A. Potash *by* Drs. E. A. Strasheim and W. A. Lombard.
Mrs. L. Fatti's son *by* Dr. G. T. du Toit.
Mrs. T. MacLeod *by* Mr. B. Murless.
Mrs. M. Ziman *by* Dr. S. Sacks.
Children of Dr. M. Ziman *by* Drs. F. Stern and G. Immerman.
Ernest Gabriel *by* Dr. P. B. Moore.
Karen Smith *by* Drs. C. Arkels and B. T. Bernstein.
Dr. B. Rogoff *by* Dr. Van der Spuy.
Dr. H. G. H. Houghton *by* Dr. H. Chait.
Clive Curson *by* Drs. L. Knox and H. Grant-Whyte.
E. C. W. H. Bergh *by* Dr. A. A. Cilliers.
Dirk Hassenkamp *by* Dr. J. B. Lurie.
Mrs. I. Wagner *by* Dr. M. M. Suzman.
Medical officers and Nursing staff of Tsumeb Corp. Ltd. *by* Various specialists.
Miss F. Stephens *by* Dr. R. R. MacKenzie.
Mrs. Z. Masey *by* Dr. H. Chait.
Family of Dr. J. N. Sher *by* Drs. Klewansky and Sieff.
Dr. W. Wilkie Scott *by* Mr. L. V. Pearson, Drs. McGinn and Grant-Whyte.
Daughter of Dr. S. Freedberg *by* Dr. R. Lang.
Dr. F. Viedge *by* Dr. Lockwood Mundy.
Mev. M. A. de Wet *deur* Dr. L. Hoogenhout, dr. en mev. D. Webster en dr. en mev. L. Krige.
Daughter of Dr. B. Epstein *by* Drs. E. Epstein, L. Melzer, M. Denny and I. Kaplan.
Dr. A. E. du Toit *by* Drs. Arklets, G. du Toit, Roux and Bersohn.
Family of Dr. J. L. Z. Njongwe *by* Drs. W. B. Fiddian-Green and J. L. Smith.
Dr. L. Bass
Dr. E. J. Dyke

Total received from Services Rendered: £172 7s. 0d.
Totaal ontvang vir Dienste Gelewer:

Donations: Skenkings:
Drs. D. M. King, G. C. Cruijwagen, J. M. Hurwitz, A. J. F. Maske, R. McDonald, C. J. S. Wiese, F. K. te Water Naude, R. Resnekov, J. S. Smit, D. S. McCall, A. S. Nethercott, T. H. Whitsitt, J. F. Rivers-Moore, G. W. Moggridge, R. C. P. Matthews, J. P. Grieve, H. E. Bernstein, F. E. Bamford, A. R. Bain, C. Akerman, C. A. V. Ovendale, F. L. Potter, J. C. Brown, R. B. Peckham, S. S. Hoffman, D. H. Hoffman, L. J. van Wyk, M. J. F. Davis, N. H. Helman, J. C. Cosnett, N. C. Hopkins, J. H. Syminton,

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	£	s.	d.
Total	88	12	6
Dr. M. J. van der Walt	3	0	0
Dr. M. G. Woolff	2	2	0
Dr. F. C. R. Smit	2	2	0
Dr. E. E. Lazarus	2	2	0
Dr. J. R. Pugh	2	2	0
Dr. C. F. Scheepers	2	2	0
Scottish Graduates Reunion, Medical Congress	17	17	0
Collection boxes at Congress	30	7	0
Bowls section of Congress Sports Committee ..	3	0	0
Dr. J. C. Jurgens	5	5	0
Natal Coastal Branch (M.A.S.A.) Womens Auxiliary: (*a*) Glamour and beauty demonstration, (*b*) Play première, (*c*) Ball, and (*d*) Braaivleis and entertainment evening	550	0	0
Medical Emergency Service Ltd.	100	0	0
Natal Coastal Branch: Vice-President's Appeal: Drs. L. Knox, L. E. Ellis, A. V. Large, A. H. Lasbrey, P. D. G. Quirke, M. L. du Toit, H. M. Barnett, D. A. S. Sichel, J. Morris, C. J. Kaplan, H. Saacks, N. R. Pooler, M. Bolton, J. S. Alexander, A. Ritten, J. Lynch, P. D. Lynch, S. Feinberg, P. B. Moore, N. Smith, J. Friedlander, H. Schaffer, D. Jacobson, T. Mulock Bentley, B. C. Murless, B. Moshal, V. Leary, D. Whitaker, J. C. Weideman, J. J. Boulle, E. M. Higgs, E. W. S. Deale, T. G. Armstrong, F. Walt, L. Atlas, S. Atlas, B. Morris, E. J. Young-Thompson, V. N. Larsen, A. Brodie, A. Bloom, A. G. Sweetapple, V. L. Pearson, G. C. Peter, M. Lipworth, W. H. H. Tanner, E. M. Barker, M. R. Gitlin, F. Stern, H. M. Pretorius, E. G. McIntosh, J. A. Hofmeyr, G. H. Gaiger, A. B. Taylor, A. Beilles, D. F. Standing, G. Gordon, M. Brien, J. Addleson, A. H. Bean, P. Pienaar, J. D. Pickford, A. D. Cuthbert, T. Dyer, H. Renton, P. Perrott, M. Renton, J. Macfadyen, R. C. J. Hill, J. G. Bickerton, H. Lazarus, C. J. Southwood, C. C. Shaw, R. Mundy, J. Booth, A. L. Ferguson, M. Byala, R. W. S. Cheetham, M. Paterson, E. W. Rayner, H. D. de Marigny, M. D. Messent ..	210	1	0
Dr. J. Knobel	2	2	0
Ladies Committee of Southern Transvaal Branch (M.A.S.A.) (Golfing account)	38	0	0
Estate late C. L. Darley-Hartley	471	13	11
Edinburgh Graduate Association	3	8	6
Cape Western Branch (M.A.S.A.) Proceeds of supper dance	119	7	0
Medical Officers of Health Group (M.A.S.A.) ..	5	5	0
Estate late Dr. L. F. B. Biccard	52	10	0
Cape Western Branch, (M.A.S.A.) Collection box	4	10	6
Natal Coastal Branch, (M.A.S.A.) Collection at Annual General Meeting	5	16	6
Total Donations: Totale Skenkings	£1,721	5	11
Grand Total: Groot Totaal	£1,928	9s.	5d.

PASSING EVENTS : IN DIE VERBYGAAN

Tak Noord-Transvaal (M.V.S.A.). Prof. en mev. I. Boerema, van Die Nederland, sal Pretoria vanaf 2 Mei tot 14 Mei besoek. Gedurende hierdie tydperk sal professor Boerema voordragte aan voor- en nagraadse mediese studente lewer. Op Dinsdag, 10 Mei, sal professor Boerema optree as gasspreker van die Tak. Sy onderwerp sal wees ‚Hipotermie en hiperpressie in die hartchirurgie'.

* * *

South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 2 May at 5.10 p.m. in the Institute Lecture Theatre. Dr. Wilbur Downs, Senior Staff Member of the Rockefeller Foundation, will speak on 'Arthropod virus'.

* * *

College of General Practitioners, Cape of Good Hope Faculty. A refresher course for general practitioners has been arranged at Rondebosch Cottage Hospital for 7 and 8 May 1960. The course will commence at 2 p.m. on Saturday 7 May. There will be two sessions—one on Saturday afternoon and another on Sunday morning. A fee of 30s. to cover the cost of the course is payable on application for enrolment. Because of restricted space in the hospital, numbers will be limited. Will those interested please communicate with the Secretary, P.O. Box 643, Cape Town as soon as possible. Further particulars are also obtainable from the Secretary.

* * *

Nutrition Society. A meeting of the British Nutrition Society will be held at Rothamsted Experimental Station, Harpenden, Herts., England, on Saturday 2 July 1960. The meeting will take the form of a Symposium on 'Food losses in field and store'. The Chairman of the meeting will be Mr. N. W. Pirie. Those practitioners who may be in Britain during July and are interested in attending this meeting may obtain further information from Dr. B. Bronte-Stewart, c/o Department of Medicine, Medical School, University of Cape Town, Observatory, Cape.

WORLD MEDICAL ASSOCIATION

INTERNATIONAL MEDICAL ADVISORY BUREAU

The Council of the British Medical Association has established an International Medical Advisory Bureau with a view to welcoming and providing a personal advisory service to medical practitioners visiting the United Kingdom. The Bureau is located at British Medical Association House, Tavistock Square, London, W.C.1.

One of the main objects of the Bureau is to welcome the overseas medical visitor, who is cordially invited to visit the Bureau as soon as possible after arrival and talk over with the Medical Director any points on which he may need advice or assistance.

Information is available at the Bureau on postgraduate education facilities and visits to hospitals and clinics can be arranged. Assistance will be given in finding suitable hotel and lodging accommodations. In addition, general information on cars, sports, travelling, exhibitions, theatres, etc. is available.

Visitors are urged to inform the Bureau of their intended visit to the United Kingdom as much in advance of their arrival as possible. Advance information as to date of arrival, mode of travel, length of stay, main objects of visit and special needs for assistance will enable the Bureau to provide the best service. Those wishing to visit hospitals or seeking advice about postgraduate courses should provide the Bureau with information as to professional experience.

All communications should be sent to: The Medical Director, International Medical Advisory Bureau, Tavistock Square, London, W.C.1., England.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

CICATRIN

Calmic, of England, announce the release from their research laboratories of a product which introduces a new concept in the treatment of infected wounds. Cicatrin consists of 2 topical antibiotics combined with 3 amino acids. The antibiotics are designed to control infection and the amino acids to stimulate wound healing.

The antibiotics contained in Cicatrin are bacitracin and neomycin, both of which are rarely used systemically. There is therefore little risk of inducing resistance or of producing sensitivity and yet Cicatrin is active against all common wound-pathogens, including penicillin-resistant strains. The 3 amino acids have been added because it has been found that they nourish and stimulate the proliferation of tissue cells, especially in cases of chronic infection where circulatory disorders have interfered with the normal supply of substances needed for the regeneration of tissue.

Cicatrin is supplied in 2 forms—as a cream and as a powder. The base of Cicatrin Cream is formulated to produce a water-in-oil emulsion when brought into contact with blood, pus or tissue exudates. The mild detergent action of the cream assists in wound cleaning, and its activity is maintained even when used in conjunction with closed-dressing technique. The active materials in Cicatrin Powder are incorporated in an absorbable starch derivative. Extremely small particle size ensures thorough dispersion over skin and tissue surfaces and the powder is useful for all conditions in which it is desirable to keep the skin dry.

Cicatrin is indicated for the treatment of both acute and chronic wounds. It is particularly suggested for varicose ulcers, burns, superficial abscesses, otitis externa, cutaneous bacterial infections, fistula in ano, etc. Cicatrin is not recommended for the treatment of fungal infections. Both Cicatrin Cream and Cicatrin Powder are supplied in 15 g. jars and stocks are available throughout South Africa. Further information may be obtained from the sole South African distributors, Messrs. Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg.

VERACTIL

Maybaker (S.A.) (Pty.) Ltd. announce the introduction of Veractil brand methotrimeprazine, and supply the following information:

Veractil shows the characteristic central properties of chlorpromazine, with additional anti-adrenaline activity and antihistamine properties comparable to those of promethazine.

The preparation is indicated in the management of psychotic illnesses and for the relief of severe pain and accompanying anxiety and as an alternative to chlorpromazine where response to the latter drug is incomplete.

Veractil is supplied in the form of varnished tablets of 5 mg., 25 mg. and 100 mg. of the acid maleate for oral administration.

BOOK REVIEWS : BOEKBESPREKINGS

PSYCHOANALYSIS

Science and Psychoanalysis. Vol. 2. *Individual and Familial Dynamics.* Edited by J. H. Masserman, M.D. Pp. vi + 218. $6.75. New York and London: Grune & Stratton, Inc. 1959.

This volume, the second of the series, again takes the form of a symposium based on the proceedings of the Academy of Psychoanalysis. The first meetings of this group, by exploring biological, philosophical and other roots of psychoanalysis, paved the way for broader reorientations, and in these papers two big questions are examined in more detail.

The first section, on masochism, opens with a frank presentation of clinical material and theoretical formulations by Dr. L. Salzman. Subsequent papers, using this as a reference, deal with particular aspects of the subject, with an emphasis on clinical considerations. The panel discussion illustrates the many diversities of opinion.

The second section deals with familial and social dynamics and perforce ranges over a wide field. Attempts to use the family as a group therapeutic unit are an example of some of the experimental work being undertaken.

This is a book which is primarily of concern to the psychotherapist, for whom there is much of considerable interest and value. W.A.S.

STRESS DISORDER

The Nature of Stress Disorder. Conference of The Society for Psychosomatic Research held at the Royal College of Physicians, May 1958. Pp. 298. Illustrations. 25s. net. London: Hutchinson Medical Publications. 1959.

This book contains, in bound form, the 15 papers read and discussed at the conference of the Society for Psychosomatic Research. The title is somewhat optimistic. Having read the book one is left feeling how great in fact is the need for research on the subject. While the papers and discussions are quite interesting, the problem of why some people break down in the face of the stresses encountered during daily life remains unsolved. Some of the papers showed an experimental approach, while others were based on clinical observations. The participants included psychiatrists, physicians and general practitioners, emphasizing the frequency of these disorders and how every doctor, whatever his sphere, has to cope with them and can contribute towards their elucidation. Hypertension, pruritus and peptic ulcer were but a few of the disorders discussed. The book makes interesting reading, but adds little to our knowledge of this important problem. I.G.

CORRESPONDENCE : BRIEWERUBRIEK

CARCINOGENIC RISKS OF IRON-DEXTRAN COMPLEX

To the Editor: In a recent Leading Article in the *British Medical Journal*[1] the carcinogenic risks of iron-dextran complex are discussed. These comments have received world-wide publicity, and their unfortunate appearance in the lay press has caused a panic amongst members of the public who have received injections of iron-dextran. The public have no acess to, and are in no position to assess, the experimental findings and scientific data on which the writer of the article concludes that, because of the carcinogenic effects of iron-dextran complex, it should be withdrawn from use in man. In all questions of carcinogenic hazard from extrinsic sources it is essential to examine and analyse critically the available facts. It is the responsibility of the medical profession, where possible, to protect the public against such hazards. This applies even more so to substances administered by the profession.

The editorial comments were based on the reported production of sarcomata in rats and mice at the site of intramuscular injection of iron-dextran complex.[2-6] Numerous other workers, who have investigated the long-term effects of iron-overload, have not recorded any malignant changes in iron-injected tissues.[7-15] The crucial practical problem arising from these animal experiments is to what extent the conclusions, especially in rats and mice, can fairly be applied to man. It is noteworthy that, although iron-dextran complex has been widely used for several years, no case of sarcoma following its use has been recorded in man.

In the applicability of these animal experiments to man, it is necessary to consider many factors. The dose of iron-dextran complex is one of the most important. Richmond[3] administered 0·4 ml. Imferon weekly for 11 - 16 months into the right upper thigh of adult male rats (a total dose varying from 17·6 to 25·6 ml., i.e. 880·0 to 1,280·0 mg. of iron) and a total of 9·5 ml. (475·0 mg. of iron) in divided doses given twice weekly over a period of 3 months into weanling rats. This dosage is massive—some 200 - 300 times the therapeutic dose in man. In the lower doses used by Richmond[3] (475 mg. of iron injected into one site), the incidence of tumours dropped sharply to become comparable with, or even lower than, published figures for tumours obtained in a similar manner by repeated injections in rats and mice of glucose, fructose, arachis oil, β-pro-piolactone, and many other innocuous materials.[16-29]

More recently, Haddow and Horning[6] injected massive doses of

iron-dextran complex subcutaneously into mice, rats, hamsters and guinea-pigs. They stated that the dosage employed by them represented, in relation to body iron, a swamping dose both locally and systemically. The 'clinical dose' for mice would be only 1/100th or 1/150th of that used in this experiment. Golberg *et al.*,[21] on the other hand, have administered repeated doses to a total of 80 times the clinical dose to rats, rabbits, mice, guinea-pigs and dogs, without any apparent effect on health. Apart from the important dosage factor, repeated trauma, muscle necrosis, and chronic inflammatory changes caused by repeated injections into the unstable subcutaneous tissues of rats and mice can lead to the production of sarcomata.[19]

Haddow and Horning[6] have stated that the injection-site sarcomata due to iron-dextran complex are 'decided primarily by the absolute amount administered and only secondarily, if at all, by the relation of this to the total-body weight'. This statement ignores the experimental work done by Beresford *et al.*[22] and Golberg,[23] who have shown that with massive doses of intramuscular iron the absorptive mechanisms are overwhelmed, residual siderophage accumulations increase and 'metabolic swamping' with iron occurs. This results locally in widespread muscle necrosis and replacement by adipose tissue and masses of siderophages and, systemically, in altered enzyme activities. The development of sarcomata may depend on massive siderotic local lesions coupled with systemic swamping of the animal with iron-dextran. In the absence of such lesions, sarcomatous change is in the highest degree unlikely to develop.[19] Indeed Haddow and Horning[6] admit that the development of sarcomata may mainly be determined by that fraction of the dose which is poorly absorbed or locally retained, and that the lesion may arise from the iron-laden connective and reticulo-endothelial tissues. In both man and animals, when iron-dextran is administered in therapeutic doses on a weight-for-weight basis, local lesions are completely absent because of rapid lymphatic removal of the majority of the injected material before local macrophage accumulation reaches appreciable proportions.[19]

Another fact worthy of comment is that sarcomata have been produced with any degree of frequency only in rats and mice. In hamsters only one sarcoma at the site of injection was produced in 82 hamsters given massive subcutaneous injections of iron-dextran complex. Rat connective-tissue cells are notoriously susceptible in responding with sarcomas to injection of the most diverse agents.[24] Other species which have received massive overdoses of iron-dextran, for example, the rabbit and guinea-pig, did not develop tumours.[6]

Other hazards have been mentioned by Nissim[25] but Golberg *et al.*[26] and MacGregor[27] have emphasized that doses of the magnitude used by Nissim[25] bear no relation to the usual total therapeutic doses in man.

Over the past few years, the efficacy of iron-dextran complex has resulted in its wide application in the treatment of iron-deficiency. This is mainly due to its ready and complete absorption from the intramuscular site. This rapid absorption in the usual therapeutic doses in man prevents local siderotic lesions and probably safeguards against the development of sarcomata. Haddow and Horning[6] admit that 'we know no more than that it is carcinogenic for the mouse and rat'. From an analysis of the available information it would appear that those who have received Imferon in the past can be reassurred about its safety.

Philip Lanzkowsky

Department of Child Health
University of Cape Town, and
Red Cross War Memorial Children's Hospital
Rondebosch, Cape
12 April 1960

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ASSISTANTS IN BENEFIT SOCIETY WORK

To the Editor: It is with regret that I noted that Federal Council indirectly decided to increase the goodwill of certain individuals' practices. I refer to the decision at the East London meeting in September 1959, that permits the incumbents of benefit society posts to use an assistant for that work.

Granted it is stated that the incumbent must do the bulk of the work. A ratio 51 : 49 would then still be a legal division of labour but hardly ethical, and it would be a very wholesale degree of 'farming-out', which it undoubtedly is. And who is to spy and prove that the proportion is not 49 : 51—or in the case of three partners—34 : 33 : 33?

The establishment of these benefit societies was primarily for the good of the very low-income groups, and not the doctors, and it could hardly have been the intention of the Medical Association to allow doctors to exploit the position in any way.

The converse trend to the benefit societies to appoint firms of partners to a single post raises some fine ethical points. Industry is now to exploit the doctors.

Feeling I might be misapprehending Federal Council's motives and decision in this matter, I wrote the Secretary for any verbatim minutes or notes he might have on this debate. I might then change my opinion or at least learn for whom not to vote at future Federal Council elections.

The reply I received stated that the Secretary was unable to let me have any notes taken at the meeting. This is an amazing reply considering there are paid stenographers at these meetings, that the meetings are open, and that the Association is apparently founded on democratic principles.

I must therefore 'willy nilly' conclude that the decision of Federal Council has no ethical basis and I must depend on hearsay in deciding for whom not to vote.

E. Epstein

409 Medical Centre
214 Jeppe Street
Johannesburg
1 April 1960

HODGKIN SE SIEKTE

Aan die Redakteur: U advies dat chemotherapie verkieslik is bo bestraling in die behandeling van Graad I limfadenoom[1] is moeilik om te begryp.

Die tumordosis wat nodig is om die primêre letsel van 'n Graad I onder beheer te bring is van die kleinste wat in radioterapie gebruik word.[2] En juis omdat hierdie dosis tot 'n klein gedeelte van die liggaam toegedien word kan 'n mens nie verstaan waarom dit 'n nadelige invloed kan hê op 'n klaarblyklike 'goeie' prognose nie. Dit sou tog meer logies wees om die murgonderdrukkende middels te bewaar vir die wydverspreide gevalle en Graad I gevalle te bestraal soos dit trouens nog algemene gebruik is.

C. L. Botha

Filkem-gebou 208
Queenstraat
Pretoria
7 April 1960

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